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DEVELOPMENT AND PREVENTION OF MYOPIA AT THE UNITED STATES NAVAL ACADEMY

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The visual standards for the line of the Navy, the branch which handles the naval vessels of the United States, have always been based on the fact that it is essential for navigational, watch and gunnery officers to have normal vision and not to have to wear glasses, which may become fogged in bad weather or may even be broken or lost, with consequent serious impairment of vision. For many years the high incidence of myopia which apparently developed among midshipmen after admission to the United States Naval Academy with supposedly normal vision was a cause of serious concern to all those interested. For years every effort was made to save such men to the service by giving them extended complete rest of their eyes before reexamination of their vision, and by retaining many of them in the Naval Academy for one to three years in the hope that their vision would improve. Every effort was also made to improve the illumination in both study rooms and classrooms for midshipmen. Decisions to retain such men were largely based on the recommendations of ophthalmologists. In the vast majority of cases, however, the vision did not improve, and the midshipman was forced to leave the naval service after two to four years in the Naval Academy. Experience showed that only about 1 per cent of such men had 20/20 vision on their final physical examination.

Prior to 1937 the visual requirement for admission to the Naval Academy was 20/20 vision in each eye unaided by lenses. The per cent of physical disqualifications of midshipmen for defective vision was so high, however, ranging from 14.14 to 31.46 per cent of the classes of 1934 to 1940 during the four year course, that it became evident that more strenuous preventive measures were necessary. Furthermore, an excessive number of junior line officers were being retired because of defective vision, and the records showed that the vision of three fourths of these had become defective (vision less than 20/20 for each eye) at the Naval Academy.

It had long been realized by the naval medical department that the chief reason for myopia developing in midshipmen was the fact that many

candidates with vision of 20/20 in each eye by the Snellen test at their physical examination for admission actually had a low degree of myopia at that time. It was evident that the time to eliminate such candidates was at the entrance physical examination. The Navy Department therefore amended the visual requirements for admission to the Naval Academy and, beginning in the summer of 1937, required all cadidates to submit to refraction of their eyes under the influence of a cycloplegic as part of their physical examination for admission. Any degree of myopia under cycloplegia, including myopic or mixed astigmatism, was made a cause for physical disqualification of the candidate even though his vision was 20/20 in each eye by the Snellen test.

The most serious objection raised to the new visual requirements was that an excessive number of candidates would be rejected on account of defective vision. Results, however, showed this objection to be unjustified. During the three years the new standard has been in effect, an average of 9.7 per cent of candidates have been rejected for defective vision, as compared with an average of 5.5 per cent during the previous four years, under the old standard. This includes the anticipated high per cent of rejections in the first year during which the new requirements were effective, high because candidates that year had not had preliminary ocular refractions to determine in advance whether they were visually qualified.

RESULTS OF USE OF NEW VISUAL STANDARD

Surveys by the United States Public Health Service have shown that the lowest incidence of refractive changes in the eyes of children occurs in the age group of 14 years and over and that approximately 40 per cent of college students have defective vision. Luckiesh stated that 21 per cent of college students show a decrease in visual acuity during the four years in college and that 14.9 per cent show a marked decrease. In spite of a visual requirement of 20/20 vision for admission to the Naval Academy, the incidence of defective vision developing among midshipmen prior to 1938 was but little less than that anticipated for college students generally, averaging 15.9 per cent for the years 1932 to 1937 inclusive, compared with the minimum 21 per cent expected for college students. The results obtained under the new visual standard have been most gratifying and fully up to expectations. The incidence of defective vision (less than 20/20 for each eye) developing at the Naval Academy in the classes of 1934 to 1943 inclusive is shown in table 1.

From these figures it is anticipated that with the present visual standard not more than 10 per cent of a class will acquire defective

vision (less than 20/20 in each eye) during their four years at the Naval Academy and that the per cent who acquire defective vision during their careers as junior line officers will be correspondingly reduced.

EXPERIENCE IN REFRACTION OF EYES OF CANDIDATES AT PHYSICAL EXAMINATION FOR ADMISSION

When refraction of eyes was commenced in the summer of 1937 with candidates for the class of 1941, the 2 per cent solution of homatropine hydrobromide commonly used for this purpose was employed. Experience quickly showed, however, that a 4 per cent solution was necessary to overcome adequately the spasm of accommodation so frequently present.

TABLE 1.—*Per Cent of Defective Vision (Less than 20/20 Each Eye) Developing Among Midshipmen from 1934 to 1940 Inclusive*

Class	Years in U. S. N. A.	Per Cent in Whom Defective Vision Developed
1934	4	16.47
1935	4	19.84
1936	4	14.14
1937	4	19.16
1938	4	20.88
1939	4	31.46
1940	4	20.80
Refraction Required on Physical Examination for Admission		
1941	3	7.03
1942	2	5.43
1943	1	1.39

In these refractions, the objective findings by retinoscopy and not the subjective findings as obtained by the use of the trial case alone are the basis for the correction of all refractive errors found. In cases of low hypermetropic and myopic errors the routine of the trial case is used in order to determine definitely the best lens combination, and this is checked with the cross cylinders. The correction is usually made for 20/15 instead of 20/20 vision in these cases because, with the exception of a few who have low hypermetropic astigmatic errors associated with some ciliary spasm, the young men all have more than 20/20 vision when their visual acuity is first taken. The candidates whose eyes cannot be corrected to vision better than 20/20 are not disqualified provided their visual acuity when not affected by induced cycloplegia is 20/20 in each eye unaided by lenses. Any candidate, however, who is found to have any degree of myopia following the use of a cycloplegic, even —0.12 or —0.25 D., is rejected. In order to be absolutely fair and to eliminate any possible error, the refractive findings for each candidate found to have myopia when the ciliary muscle is under the

influence of a cycloplegic, are checked by two other specialists on the examining board, and the candidate, prior to final rejection, is then completely reexamined visually by another medical board with an entirely different personnel from the first board.

NECESSITY OF A HYPEROPIC RESERVE

Review of reports of refraction of the eyes of candidates at the time of a preliminary physical examination and of the refractive findings in the same cases at the time of physical examination for admission, six to fifteen months later, has shown that the great majority of candidates whose refraction was of the plano type, or 0.25 D. of hypermetropia, at the time of the preliminary physical examination and who were therefore physically qualified at that time showed a low degree of myopia at the time of their entrance physical examination and were therefore rejected. Occasionally a candidate who showed as much as 0.5 D. of hypermetropia on preliminary examination would be found to have 0.25 D. of myopia at the time of physical examination for admission. The vast majority of candidates whose refraction was of the plano type, or 0.25 D. of hypermetropia, on their entrance examination and some who showed 0.5 D. of hypermetropia at this time and were therefore physically qualified for admission were found to be physically disqualified because of defective vision (less than 20/20 in each eye) within one or two years after admission to the Naval Academy. During the interval, these candidates had all acquired approximately 0.5 D. of myopia. These changes in refraction, while partly due to natural causes (age), were evidently largely caused by the intensive study which these young men performed during the intervening time. As is well known, an emmetropic eye is for practical purposes an abnormal eye, the great majority of persons with so-called normal vision being actually hypermetropic. Those candidates, then, whose refraction was of the plano type had borderline conditions definitely on the way to myopia. For all practical purposes, experience here has shown that patients with 0.25 D. of hypermetropia are in the same class.

In view of the experience at the Naval Academy during the past three years, as described, it is evident that a reserve of preferably 1 D. and at least 0.5 D. of hypermetropia is necessary at the time of preliminary refraction to be reasonably sure that the candidate will pass his physical examination for admission. Furthermore, in order to be reasonably sure of being visually qualified for a commission in the line of the Navy after four years at the Naval Academy, it is necessary for the midshipman to have a hyperopic reserve of at least 1 D. of hypermetropia at the time of admission. Of course, an occasional candidate with 0.25 to 0.5 D. of hypermetropia at the time of admission will survive visually and receive a commission, but this is exceptional.

In this connection, it should be stated that the visual standard for retention in the Naval Academy and for commission in the line of the Navy is 20/20 vision in each eye unaided by lenses, except for eyes shown by refraction to be hypermetropic, when vision of 18/20 is allowed. Midshipmen are not visually disqualified because of evidence of myopia under the influence of a cycloplegic but only if they have vision of less than 20/20 in each eye by the Snellen test. Furthermore, it must be emphasized that midshipmen failing to make 20/20 by the Snellen test at any annual physical examination are given every consideration, such as complete rest of the eyes for a reasonable time and several visual reexaminations at different times by different medical examining boards, before being finally found physically disqualified because of deficient vision.

ATROPINE AS A CYCLOPLEGIC

Shortly after refraction was introduced as a part of the physical examination for admission (in 1937) a prominent civilian ophthalmologist invited the attention of the Navy Department to the fact that a candidate who had been rejected because of 0.25 D. of myopia in each eye under homatropine cycloplegia showed 0.25 D. of hypermetropia when atropine was used as a cycloplegic. It was argued that such a candidate should be accepted because he actually showed no evidence of myopia under a cycloplegic (atropine sulfate). As there were no reliable statistics at that time to indicate the visual prognosis in such cases, the Navy Department directed that the candidate in question be accepted and that other candidates showing not more than 0.25 D. of myopia under the influence of homatropine should be tested with atropine as a cycloplegic, those showing no evidence of myopia under atropine cycloplegia to be accepted. This procedure was followed during the summers of 1937 and 1938 with candidates for the classes of 1941 and 1942.

While the naval medical department anticipated that a considerable number of these candidates would be found visually disqualified before they completed four years at the Naval Academy, it was felt that, in the absence of definite information to the contrary and in fairness to the candidates involved, they should be given the benefit of the doubt.

The findings following use of atropine as a cycloplegic in these cases were most interesting. It was anticipated that few men showing 0.25 D. of myopia with 4 per cent homatropine hydrobromide used as a cycloplegic would show no evidence of myopia under the influence of atropine. Actually, 127 such candidates were tested and 64.54 per cent were accepted. The visual careers of these candidates as midshipmen are shown in table 2.

It will be noted from table 2 that the "visual mortality" of candidates tested with atropine was more rapid in the class of 1942 than in the class of 1941. No special reason for this has been discovered. However, after two years' experience with borderline visual conditions in men admitted after refraction with atropine as a cycloplegic, the Navy Department decided that "visual mortality" was so high the use of atropine was definitely not worth continuing. Its use as a cycloplegic was therefore discontinued, and all candidates for the class of 1943, physically examined for admission in the summer of 1939, who showed any evidence of myopia under cycloplegia induced with 4 per cent homatropine hydrobromide were rejected.

TABLE 2.—*Visual History for Three and Two Years of Candidates Tested by Refraction Under Atropine*

	Class of 1941	
(*a*)	Number tested with atropine	66
(*b*)	Per cent accepted	65.15
(*c*)	Per cent of (*b*) visually disqualified (vision less than 20/20 each eye) first year	13.33
(*d*)	Per cent of (*b*) visually disqualified (vision less than 20/20 each eye) second year	20.00
(*e*)	Per cent of (*b*) visually disqualified (vision less than 20/20 each eye) after three years	36.66
(*f*)	Total per cent of class on admission (569) showing defective vision (less than 20/20 each eye) after three years as midshipmen	7.03
(*g*)	Per cent of (*f*) admitted after refraction with atropine	27.50
	Class of 1942	
(*a*)	Number tested with atropine	61
(*b*)	Per cent accepted	63.93
(*c*)	Per cent of (*b*) visually disqualified (vision less than 20/20 each eye) first year	33.33
(*d*)	Per cent of (*b*) visually disqualified (vision less than 20/20 each eye) after two years	52.77
(*e*)	Total per cent of class on admission (685) showing defective vision (less than 20/20 each eye)) after two years	9.43
(*f*)	Per cent of (*e*) admitted after refraction with atropine	46.34
	Classes of 1941 and 1942	
	Per cent of all acceptances after refraction with atropine remaining in the Naval Academy after three and two years, respectively *	26.77

* Fifteen men tested with atropine cycloplegia failed scholastically and are not included in this table except for (*a*) and (*b*).

INVESTIGATIONS OF ILLUMINATION FOR STUDY PURPOSES

During past years, criticisms of the illumination provided at the Naval Academy for study purposes have been made from time to time, it being alleged that faulty illumination was the cause of the high incidence of myopia developing among midshipmen. While the medical department realized that the incidence of myopia developing at the Naval Academy was less than that noted in civilian universities, the necessity for good illumination as an aid in the preservation of normal vision was

recognized. Every effort was made to provide adequate illumination for study and for classroom purposes. The last extensive research along these lines was made by a board of nationally prominent illuminating engineers in 1933-1934. Among various improvements then recommended were the standard I. E. S. (Illuminating Engineers Society) table study lamps now used by midshipmen. These lamps use a 100 watt bulb, provide semi-indirect illumination and are placed in the center of the study table. Their installation was completed during 1936. While these lamps were much more efficient than the old, their continued use has demonstrated that they are not entirely satisfactory to either medical officers or midshipmen, because of a certain amount of both direct and specular glare. Furthermore, the upturned bowls of these lamps are definite dust collectors, and the dust frequently decreases the foot-candles on the working surface. During the past year, therefore, further investigations have been made in an endeavor to obtain a more satisfactory table study lamp. It was found that a recently developed table lamp of the fluorescent, or daylight, type was apparently the best. Six trial lamps of this type were therefore obtained and installed during the summer of 1939. The eyes of the midshipmen using these lamps and those of a control group using the standard lamp were carefully checked by refraction, all having approximately the same refractive errors. These trial lamps will be used for a year, after which the eyes of the midshipmen concerned will again be carefully checked by refraction. While the results will of course not be conclusive, they will at least be a fair indication of what may be expected concerning the effect of these lamps on the maintenance of normal vision.

These trial lamps are clamped to the side of the study table. They are so constructed that they may be turned to the position most suitable for use when the student is writing on the table or reading beside the table. The advantages of these lamps are:

(*a*) The light is steady and of the daylight type, the most efficient artificial illumination, and is preferred by most eyes for prolonged and critical visual work.

(*b*) Illumination is adequate but not too bright, varying from 20 to 28 foot-candles on the working surface.

(*c*) The visual object or task is the brightest object in the room. With the simultaneous use of a 100 watt indirect ceiling light, the general illumination in the room is adequate to relieve sharp contrasts, 2.5 to 3 foot-candles on the walls, but the table lamp is not so bright as to distract the eyes from their task.

(*d*) The direction of light from its source is good, from the left for right-handed persons and from the right for left-handed persons.

(*e*) Glare is completely absent.

(*f*) The operating cost is low. The cost of operation of two of these fluorescent lamps, one for each student in a two man room, is slightly less than that of using the 100 watt bulb required in the present standard lamp.

PRESENT VISUAL STANDARDS FOR MIDSHIPMEN AND FOR COMMISSION IN THE NAVY

Many letters are received by the Naval Academy authorities from both civilian ophthalmologists and laymen requesting information about the visual requirements for admission to the Naval Academy, for retention as midshipmen and for commission in the Navy. The regulations covering these points are therefore quoted in the following paragraphs:

(*a*) For admission to the Naval Academy.

Each candidate on entrance to the Naval Academy must have normal or 20/20 vision in each eye and must submit to refraction under a cycloplegic. Any degree of myopia or myopic astigmatism shall cause rejection of the candidate.

(*b*) For retention in the Naval Academy.

(1) Fourth [Freshman] and Third [Sophomore] Classes.

The visual standard for each member of the Third and Fourth Classes shall be 20/20 each eye in the presence of myopia or myopic astigmatism or 18/20 each eye without myopia or myopic astigmatism.

Those who fail to meet these standards shall be brought before a Board of Medical Survey with a view to their discharge from the Naval Service.

(2) Second (Junior) Class.

Midshipmen having 18/20-20/20 vision, with or without myopia, will be recommended for retention at the Naval Academy. In the cases of those having 15/20-18/20 vision, special reports will be made to the Bureau of Medicine and Surgery for opinion prior to final action.

Those midshipmen having less than 15/20 vision shall be brought before a Board of Medical Survey with a view to their discharge from the Naval Service.

(3) First (Senior) Class.

Those having 18/20-20/20 vision without myopia will be recommended for full line commission.

Those having 18/20-20/20 vision with myopia will be recommended for a revocable commission for a period of two (2) years, to be followed by a physical examination at the end of that time to determine their fitness for a regular commission in the line or staff of the Navy.

Those having 15/20-18/20 vision will be recommended for a revocable commission for a period of two (2) years, to be followed by a physical examination at the end of that time to determine their fitness for a regular commission in the staff corps of the Navy.

Those members of the Graduating Class of the Naval Academy having less than 15/20 vision, each eye, will be brought before a Board of Medical Survey with a view to their discharge from the Naval Service.

In aviation, more than 1 D. of hypermetropia is disqualifying, because as the aviator grows older, his vision will become less than 20/20, and,

further, since accommodation is necessary at all times for good distance vision, eyestrain and eventually some ciliary spasm will occur, with resulting temporary impairment of distance vision which may result in the "cracking up" of the plane, especially on landing.

CONCLUSIONS

It is considered that during the past three years the Naval Academy has definitely proved the necessity of midshipmen having a hyperopic reserve of at least 1 D. at the time of admission to the Naval Academy and of their meeting the present visual requirements if the visual standards of the line of the Navy are to be maintained.

Results following the requirement that refraction under the influence of a cycloplegic be made a part of the physical examination for admission, with rejection of those showing any evidence of myopia under the induced cycloplegia, have shown that an unduly high percentage of candidates are not eliminated and that the development of myopia among midshipmen has been considerably reduced. It is anticipated that the percentage of candidates rejected to date under the present visual standard will be somewhat reduced as the visual requirements become more widely known and understood.

It is desired to invite especially the attention of ophthalmologists to the importance of this subject. They can save many patients much time, worry and money by understanding the visual requirements of the Naval Academy and by advising candidates coming to them for preliminary ocular refraction definitely as to their visual prospects, so far as a naval career is concerned. Any candidate having less than 0.5 D. of hypermetropia at the time of a preliminary ocular refraction should be informed that, while visually qualified at that time, he has a borderline condition which may progress to a low degree of myopia by the time he takes his physical examination for admission to the Naval Academy and may therefore be rejected. Any candidate having from 0.5 to 1 D. of hypermetropia at the time of a preliminary ocular refraction should be informed that, while he should pass the physical examination for entrance to the Naval Academy, he stands no better than an even chance visually of obtaining a commission in the line of the Navy on graduation.

TREATMENT OF THROMBOSIS IN THE CENTRAL VEIN OF THE RETINA WITH HEPARIN

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Previous to the introduction of heparin it had been discovered that certain mammalian tissues contain one or more anticoagulants. McLean,[1] in Howell's laboratory in 1916, first isolated an active fraction which in 1918 Howell and Holt [2] named heparin and which was at first isolated exclusively from the liver tissue of the dog. Later, Charles and Scott [3] showed that this substance is present in varying amounts in the lung, liver and skeletal muscles of the ox, the lung containing the largest amount. During the past five years the anticoagulant properties of heparin have been available for use outside the laboratory, owing to the work of Best [4] and his co-workers in Toronto, Canada, and also to that of Jorpes and his school [5] in Stockholm, Sweden. Jorpes described heparin as a polysaccharide of high molecular weight which also contains nitrogen and sulfur. The purest preparation contains 40 per cent of sulfuric acid. Best [4] found that as the result of purification a crystalline barium salt of uniform potency was obtained. An amorphous benzidine compound was also secured which had the same empiric formula ($C_{25}H_{65}O\ 50\ N_2S_5$). Charles and Scott [3] have shown that when the crystalline salt is treated with excess of ammonium carbonate solution at 65 C. the barium may be completely removed. The heparin is then precipitated with acetic acid, centrifuged out, washed with ether and dried. It can be stored as a dry powder. From this when required a solution can be made. Tricresol may be used and the solution sterilized by passing through a Berkefeld or a Zeitz filter. A short time ago the

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1. McLean, J.: The Thromboplastic Action of Cephalin, Am. J. Physiol. **41**: 250, 1916.

2. Howell, W. H., and Holt, E.: Two New Factors in Blood Coagulation—Heparin and Pro-Antithrombin, Am. J. Physiol. **47**:328, 1918.

3. Charles, A. F., and Scott, D. A.: The Preparation of Heparin, J. Biol. Chem. **102**:425 and 431, 1933; Tr. Roy. Soc. Canada **28**:55, 1934.

4. Best, C. H.: Heparin and Thrombosis, Brit. M. J. **2**:977, 1938.

5. Jorpes, E.: The Chemistry of Heparin, Biochem. J. **29**:1817, 1935. Jorpes, E.; Holmgren, H., and Wilander, O.: Ueber das Vorkommen von Heparin in den Gefässwänden und in den Augen, Ztschr. f. mikr.-anat. Forsch. **4**:179, 1937.

work of Dam and Glavind [6] indicated that the hemorrhagic tendency observed in cases of jaundice and disease of the liver is due to prothrombin deficiency and can be repaired by administration of the fat-soluble vitamin K. Prothrombin may be looked on as a ferment precursor which under the influence of calcium and tissue extract is converted into the ferment thrombin; thrombin acting on fibrinogen produces a clot, or fibrin. The conversion of prothrombin into thrombin and the action of the latter on fibrinogen can be inhibited by heparin.

If a single dose of heparin is given intravenously the clotting time becomes prolonged, the increase being proportionate to the size of the dose. Best pointed out that there is no negative phase; that is, the clotting time does not go below normal after a massive dose of heparin. It comes back fairly accurately to the initial value.

An interesting experiment was made by Solandt and Best.[7] They dissected out the coronary artery of a dog for ½ inch (1.2 cm.) or so and clamped it at either end. Then they introduced sodium ricinoleate into the lumen of the vessel, and after five minutes thrombi were formed, owing to the irritation produced. When the animals were heparinized before the injection of sodium ricinoleate, however, thrombi formed in only 1. Here, as Best pointed out, is a suggestion as to the potential usefulness of heparin in the treatment of coronary thrombosis.

Heparin can be used in blood transfusion either by adding it to the blood as it is collected or by injecting it into the donor, giving 1 mg. of heparin per kilogram of body weight, the blood being withdrawn one to five minutes later. At the end of one and one-half to two hours the coagulation time of the donor's blood returns to normal.

In general surgical practice it is in operations on the vascular system that heparin is of the greatest use. Murray and Best [8] have carried out the removal of clot from the interior of vessels, and by heparinizing the patient time is given (by preventing clotting) for the intima of the vessel to heal. In those major abdominal operations in which embolism and thrombosis are peculiarly common heparin has been used most successfully, and probably in wartime surgical practice, as has been suggested by Lockwood, it will be most useful.

Heparin is the most active of all anticoagulants; it is nontoxic, and its use in no way interferes with analysis of the blood.

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6. Dam, H.: Vitamin K, Ztschr. f. Vitaminforsch. **8**:248, 1938. Dam, H., and Glavind, J.: Clotting Power of Human and Mammalian Blood, Acta med. Scandinav. **96**:108, 1938.

7. Solandt, D. Y., and Best, C. H.: Heparin and Coronary Thrombosis in Experimental Animals, Lancet **2**:130, 1938.

8. Murray, D. W. G., and Best, C. H.: Heparin and Thrombosis, J. A. M. A. **110**:118 (Jan. 8) 1938.

In the *Lancet* in 1938, Holman and Ploman,[9] of Stockholm, Sweden, reported the case of a nurse aged 52 years with partial thrombosis in the main trunk of the central vein of the retina. She was treated with heparin and recovered rapidly and completely. In the same issue Magnusson[10] reported from the Maria Hospital (in Stockholm) a case of thrombosis in the posterior inferior cerebellar artery, a so-called Wallenberg syndrome,[11] which, treated with heparin, was cured.

Venous thrombosis affects the central vein of the retina or one of its branches; according to Parsons,[12] the obstruction in the central vein is always just behind the lamina cribrosa. The ophthalmoscopic picture of the retina is well known: swollen and tortuous veins; blood escaping from the capillaries and forming innumerable hemorrhagic deposits over the retina, and pronounced stasis phenomena in and around the papilla. Secondary glaucoma in some cases follows in a few months, but not so commonly as textbooks would lead one to suppose, although in an eye with a disposition to glaucoma thrombosis may easily cause increased tension. I have trephined some such eyes with secondary glaucoma and have saved the eyeball from being removed.

TREATMENT WITH HEPARIN

Last year my friend Dr. K. G. Ploman, of Stockholm, sent me details of 8 cases of thrombosis treated with heparin. In 2 of these cases the thrombi were in the trunk, and in 6 they were in the branches, of the central vein. His results were as follows: In 1 case of thrombosis of the trunk complete recovery took place within a short time, and 5 of the 6 patients with branch thromboses rapidly presented favorable improvement of vision. The average age of the patients was 58 years. He compared these results most favorably with those observed in former cases, in which the patients were treated with potassium iodide.

I have treated 5 patients during the past year, 3 of whom had complete trunk thromboses, and 2, branch thromboses. In only 1 instance of complete trunk thrombosis did complete failure follow, the eye becoming glaucomatous and blind. In the remaining 2 cases some improvement was evident. In a man aged 58 the vision of the eye rose from 2/60 to 6/24 partly. In the 2 patients with branch thromboses complete cure resulted, central vision in each being 6/6. A woman aged 56 had noticed

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9. Holman, N., and Ploman, K. G.: Thrombosis of Central Vein of Retina Treated with Heparin, Lancet **1**:664, 1938.

10. Magnusson, J. H.: Thrombosis of the Posterior-Inferior Cerebellar Artery (Wallenberg Syndrome) Treated with Heparin, Lancet **1**:666, 1938.

11. Wallenberg, A.: Acute Bulbäraffection, Arch. f. Psychiat. u. Neurol. **27**: 504, 1895; **34**:1923, 1901.

12. Parsons, J. H.: Venous Thrombosis, in Diseases of the Eye, ed. 9, New York, The Macmillan Company, 1938, p. 357.

a slight headache the previous day, but on the morning of her visit to me there was dimness of vision of the left eye. The vision actually was 6/36. The condition turned out to be thrombosis of the inferior branch of the central vein of the left eye. On the same day I was able to begin the heparin treatment. Two hundred and fifty milligrams per day was given in two injections. This is equivalent to 5 cc. of a 5 per cent solution, and such a solution is made up in a small vial ready for use. Altogether this patient received 30 cc., or the contents of six small bottles. Untoward symptoms were not experienced until the fifth and sixth injections were given. These were followed by flashes of violet light in both eyes. The vision rose from 6/36 on July 21 to 6/6 on October 21 and has remained the same since, the retina being absolutely free from hemorrhages.

Ploman [13] asked: "How can heparin affect a venous thrombosis?" and he suggested that the coagulation of the blood is retarded; the blood becomes more fluid; new fibrin is prevented from forming on the thrombus, and loose blood corpuscles are washed away, so that the thrombus does not become complete. The heparin permeates into the capillaries and may contribute to the more rapid absorption of the extravasated hemorrhages. Apparently Ploman has found, just as I have, that when patients present themselves some days after the onset of thrombosis the results are not good but when treatment begins the same day or immediately afterward good results are obtained.

Heparin can also be used for extensive hemorrhages into the retina or the vitreous. The cost of the product is the greatest obstacle to its use. The 5 cc. bottle costs 33 shillings ($8). It is not necessary to keep the patient in bed all the time. Ambulatory patients can have two injections daily, each of 100 to 125 mg., making a total dose, say, of 250 mg., although Ploman frequently gave injections every four hours up to 400 mg. daily. This amount, however, was given only to patients in bed. The needle should not be too small, and when injection into the vein is made slight pressure should be applied to prevent the escape of noncoagulable blood into the subcutaneous tissues.

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13. Ploman, K. G.: Heparin Treatment of Thrombosis in the Central Vein of the Retina, Acta ophth. **16**:502, 1938.

SOME PRACTICAL CONSIDERATIONS PERTAINING TO CORNEAL EDEMA

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Recent work concerning the experimental production of bullous keratitis has resulted in at least two important conclusions:[1] First, the edema fluid in this condition is derived from tears or from some fluid used as a substitute on the outside of the eye. Second, for the production of experimental corneal vesiculation the osmotic pressure of the fluid within the cornea must be greater than that on the corneal surface. From these conclusions have come several discoveries of practical value. Those relating to the therapy of bullous keratitis, together with a description of the pathologic changes in experimental bullous keratitis, will be reported later. There are, however, several incidental observations which would not be appropriately included under the heading of bullous keratitis but which nevertheless have considerable clinical value and may therefore be separately reported at this time.

The first observation to be reported concerns the so-called toxic action on the corneal epithelium of cocaine. As is well known, the use of a few drops of cocaine hydrochloride solution on the eye will result in apparent drying of the corneal surface, provided the eye is kept open. The cornea under these conditions loses its customary luster, and a drop of water placed on the eye will remain intact, as it will do on any dry surface. With the slit lamp and the microscope it is apparent, however, that despite this drying of the surface there is actual edema of the epithelium. It is as though the epithelium had imbibed what fluid was available and thus had left the surface dry. This edema produced by cocaine is so similar to that seen in the initial stages of experimental bullous keratitis that I suspected that it, too, might be due to hypotonicity of the fluid on the outside of the eye. This seemed all the more likely in view of the fact that cocaine produces no corneal edema when the lids are kept closed and the surface of the cornea is thereby kept moist with tears. Accordingly, I had a solution of cocaine hydrochloride made up of the strength (4 per cent) customarily employed for routine cocaine anesthesia but added to it sufficient sodium chloride to make

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1. Cogan, D. G.: The Experimental Production of So-Called Bullous Keratitis, Arch. Ophth., to be published.

the solution isotonic with 1.5 per cent salt solution. I chose the 1.5 per cent concentration because such a solution is approximately the reported osmotic equivalent of tears and because, as was noted previously, tears prevent the corneal changes. When this new cocaine solution was used in one eye and the usual cocaine solution in the other eye, the difference in the two corneas was striking, attesting the correctness of the aforementioned hypothesis. With the new solution there was practically no drying of the surface or edema of the epithelium.

The discovery that hypotonicity of the solution used is responsible for much of the alleged toxic action of cocaine accounts also for other observations which have heretofore seemed paradoxic. Thus, cocaine hydrochloride in 10 per cent solution usually produces less effect on the corneal surface than does a 4 per cent solution. This is easily observed by instilling a 4 per cent solution into one eye of a rabbit and a 10 per cent solution into the other eye. After a few minutes the eye into which the 4 per cent solution has been instilled will show considerable irregularity of the surface and edema of the epithelium. The eye also assumes a characteristically lusterless and dry appearance. The eye into which the 10 per cent solution has been instilled shows these changes to a much less degree, if at all. Here the so-called "toxic" effect of cocaine is the reverse of what one would expect on the basis of toxicity, but it is consistent with the differences in osmotic pressure of the two solutions.

A second fact that has come to light during these investigations concerns the corneal clouding noted especially during operations for separated retina. Any one who attempts to make ophthalmoscopic examinations during such operations knows how annoying this may be.

As the experience at the Massachusetts Eye and Ear Infirmary in this regard may differ from that elsewhere, I shall first describe the routine procedures used in that institution and then mention what the difficulty in this connection has been and how it may be overcome.

It has been the custom here, in doing operations for separated retina, to use pontocaine in preference to cocaine. For preoperative irrigation of the conjunctival sac and for keeping the cornea moist during the operation a solution half the strength of physiologic solution of sodium chloride, that is, about 0.4 per cent, is used. This hypotonic solution has been used at the Massachusetts Eye and Ear Infirmary since Reese's observation [2] that it caused less striate keratitis than did physiologic solution of sodium chloride. However, Reese's observations were concerned only with the effects of intraocular irrigation, and, as my work on bullous keratitis has shown, there are different effects when hypotonic solutions are used on the outside of the eye and when they are used intraocularly. In any case the corneal epithelium has invariably

2. Reese, R.: Personal communication to Dr. F. H. Verhoeff, about 1925.

become cloudy during the procedure of irrigating with hypotonic saline solution for prolonged operations. Although the clouding is never marked, it is enough to handicap use of the ophthalmoscope, and I have particularly desired a clear cornea since adopting Weve's method of localizing the hole during the operation. As the changes in the corneal epithelium are similar to those due to hypotonicity of cocaine hydrochloride solutions, described in the foregoing paragraphs, and as covering the cornea with the lids prevents the epithelial changes in both instances, it seemed reasonable to suppose that the clouding during operations for separated retina is also due to relative hypotonicity of the fluid outside the eye. I therefore substituted a 1.5 per cent solution of sodium chloride as an irrigating fluid for the 0.4 per cent solution, and for the first time had the satisfaction of having a clear cornea throughout the entire operation. Even when the eye became temporarily glaucomatous, as it almost invariably does when sufficient surface diathermy is used, the cornea remained clear. It is possible that corneal clouding during operations for separated retina has not been so troublesome for those using physiologic solution of sodium chloride instead of the half strength solution, but I should certainly expect some epithelial changes, as physiologic solution of sodium chloride is hypotonic to corneal fluid.

One can strikingly demonstrate the effect of different osmotic pressures by performing the following simple experiment: Instill a 4 per cent solution of cocaine hydrochloride into both eyes of a rabbit two or three times and then keep one cornea moist with distilled water and the other cornea moist with 3 per cent saline solution. Within a few minutes the cornea exposed to the distilled water will become gray and lusterless, while the other cornea will remain essentially normal. If stronger salt solutions are used, however, there may be a late effect, coming on ten minutes or so after the irrigation has been stopped, at which time the cornea exposed to the hypertonic salt solution becomes slightly grayish. This is undoubtedly due to the fact that the cornea has absorbed sufficient salt from the irrigating fluid to be hypertonic to the tears.

A third observation on corneal edema clarifies the etiology and the treatment of the clouding that occurs as a result of the use of contact glasses. I was led to investigate this by a suggestion of Dr. F. H. Verhoeff that the clouding might result from a difference in osmotic pressure similar to that which causes corneal edema in experimental bullous keratitis. I investigated this subject by wearing before one eye a contact glass with the usual solution of sodium chloride of half physiologic strength (0.4 per cent) and before the other eye a contact glass with a 1.5 per cent solution. There was no gross difference between the corneas until approximately three quarters of an hour had elapsed. Then the cornea which had been in contact with the 0.4 per

cent solution of sodium chloride became slightly cloudy, while the other cornea remained clear. At the end of one and one-half hours the difference was more marked. So far as could be judged grossly and by slit lamp examination, the cornea which had been in contact with the 1.5 per cent solution of sodium chloride showed no change at any time.

At the end of one and one-half hours the contact glasses were removed. The vision of the eye which had been exposed to the 0.4 per cent solution of sodium chloride was slightly blurred, and a conspicuous rainbow, with red outermost and blue innermost, was present about lights. The rainbow, which reminds one of similar complaints by glaucomatous patients, gradually diminished in size and intensity but nevertheless was still visible as late as an hour and a half after the contact glasses had been removed. Immediately after the contact glass was removed from this eye there was some edema of the epithelium, detectable with the slit lamp. But it was of interest to me that even when the colored rings about the lights were subjectively very striking the objective edema of the epithelium was slight.

The vision of the eye which had been exposed to the 1.5 per cent solution of sodium chloride, however, was at no time impaired by blurring, and no trace of rainbows about lights could be detected before or after the removal of the contact glass.[3]

This observation with contact glasses has undoubtedly many analogies which will become evident in the course of time. I might mention here, however, an observation which was called to my attention by Dr. Trygve Gundersen. If one swims under fresh water with the eyes open for any length of time, one may subsequently see colored rings about lights and actual edema of the cornea may be detectable with the slit lamp. One may verify this by the following simple experiment: Bathe the cornea for fifteen to twenty minutes in distilled water (with an eye cup); then note the colored rings about lights. With the slit lamp there will be seen slight epithelial edema. If a 1.5 per cent solution of sodium chloride is used instead of distilled water, no such phenomenon occurs. It is interesting that the colored rings cannot be seen until the distilled water has been used for ten to fifteen minutes and that they then persist for about fifteen minutes after discontinuance of the eye bath.

3. The fact that hypertonic saline solution has not been used generally with contact glasses is perhaps due to the fear of what hypertonic concentrations would do to the cornea. Thus, Salvatori (P. S. Salvatori, Obrig Laboratories, Inc., 7 East Forty-Eighth Street, New York) in his handbook on contact glasses, said that hypertonic solutions cause a "dehydration" of the cornea. I do not know what the evidence for this is, but for other corneal conditions I have used concentrations of 3 per cent sodium chloride over a period of several months and have never found the slightest deleterious effect from it.

A fourth observation of practical value which is appropriately considered under the heading of corneal edema pertains to ophthalmoscopy for acute glaucoma. If the cornea is too steamy or edematous to permit a clear view of the fundus, one may temporarily reduce the hindrance by the simple expedient of bathing the eye in hypertonic salt solution. For this I first used a 3 per cent sodium chloride solution, but, as this took fifteen to twenty minutes for clearing, I now employ a 10 per cent solution in an eye cup. The cornea shows definite clearing in a few minutes. The explanation is fundamentally no different from that for any of the foregoing observations.

SUMMARY AND CONCLUSIONS

It has been previously shown that corneal edema may result from a relative hypotonicity of the fluid bathing the cornea. The purpose of this paper is to report several observations of practical value arising from this conclusion.

The change occurring on the surface of the cornea when cocaine hydrochloride is used is due largely to the hypotonicity of the solution used rather than to the direct toxic action of cocaine itself, as is generally believed. The cocaine-produced diminution in the blink reflex probably contributes to the development of these changes in the cornea. When sufficient sodium chloride is added to the cocaine hydrochloride solution to make it approximately isotonic with tears, the corneal changes are practically eliminated.

The opacity of the cornea that occurs during certain procedures, notably operations for separated retina, in which prolonged irrigation of the surface of the cornea is involved is due largely to hypotonicity of the irrigating fluid. The opacity, which may seriously handicap ophthalmoscopic examination, is effectively eliminated by the use of a 1.5 per cent solution of sodium chloride for the irrigating fluid instead of the usual weaker concentrations.

The corneal opacity which commonly occurs with the use of contact glasses is similarly due to hypotonicity of the fluid used and may be eliminated by the substitution of a 1.5 per cent solution of sodium chloride for weaker solutions. Although the observations reported here pertain to the type of contact glass used as a substitute for ordinary glasses, the conclusions would also apply to the type of contact glass used in gonioscopic examination.

The corneal edema which occurs with acute glaucoma and prevents adequate ophthalmoscopic study may be greatly diminished by bathing the cornea in hypertonic saline solution for a few minutes.

OCULAR FINDINGS ASSOCIATED WITH DYSOSTOSIS MULTIPLEX AND MORQUIO'S DISEASE

REPORT OF A CASE OF THE FORMER

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Dysostosis multiplex was first reported by Hurler [1] in 1919 but was given its present name by Binswanger and Ullrich [2] in 1933. There are about 25 published cases. This rare form of dwarfism, usually discovered during the first year of life, is characterized by scaphocephalic or chondrodystrophic deformities of the skull and by chondrodystrophic changes in other parts of the skeleton. The head is large and the facial portion of the skull massive, with prominent frontal bosses. The root of the nose is broad and depressed. The alveolar processes are large and the tongue thick and fleshy. The neck is so short as to appear almost nonexistent. The trunk is short, the spine kyphotic and the abdomen protuberant. The liver and the spleen are enlarged. The limbs are short and plump, with limitation of movements of the joints. As a rule the mentality is impaired. In all the reported cases there was diffuse interstitial corneal clouding, usually with other ocular abnormalities. The corneal haze is not due to elevated intraocular tension and is unaffected by miotics, mydriatics and surgical reduction of pressure. In 1 instance [3] microscopic examination of the eye showed separation of the lamellas of the cornea but otherwise revealed no abnormality. By special staining, Berliner [4] was able in his case to demonstrate a lipoid substance in these spaces. In both cases autopsy showed diffuse evidence of lipoidosis. In another instance the authors [5] com-

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Read at the meeting of the Pacific Coast Oto-Ophthalmological Society, Spokane, Wash., June 26, 1940.

1. Hurler, G.: Ueber einen Typ multipler Abartungen, vorwiegend am Skelettsystem, Ztschr. f. Kinderh. **24**:220-234, 1920.

2. Binswanger, E., and Ullrich, O.: Ueber die "Dysostosis multiplex (Typus Hurler) und ihre Beziehungen zu anderen Konstitutionsanomalien, Ztschr. f. Kinderh. **54**:699-712, 1933.

3. Kressler, R. J., and Aegerter, E. E.: Hurler's Syndrome (Gargoylism), J. Pediat. **12**:579-591 (May) 1938.

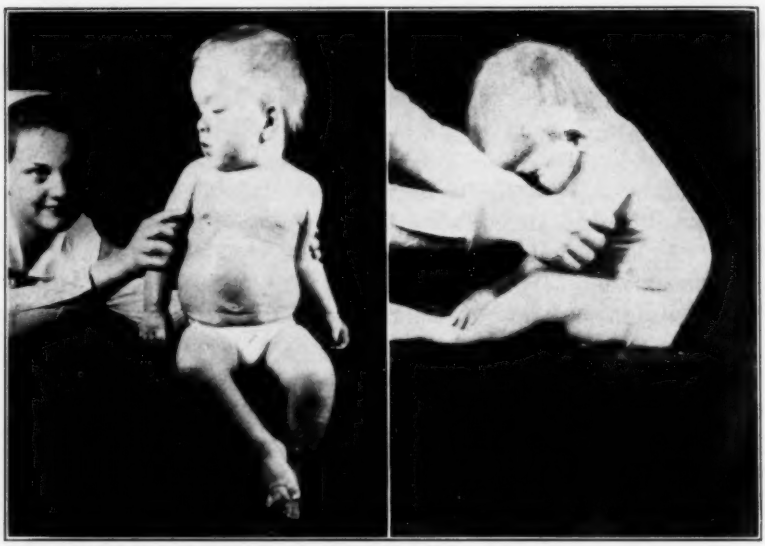
4. Berliner, M. L.: Lipin Keratitis of Hurler's Syndrome (Gargoylism or Dysostosis Multiplex), Arch. Ophth. **22**:97-105 (July) 1939.

5. Slot, G., and Burgess, G. L.: Gargoylism (Case), Proc. Roy. Soc. Med. **31**:1113-1116 (July) 1938.

mented that there was undoubtedly grave disturbance of lipoid metabolism. There is no hereditary factor.

REPORT OF CASE

My own patient (D. W.) was first seen in March 1939, in the Spokane unit of the Shriners' Hospital for Crippled Children, at which time he was 18 months of age. His resemblance to the photographs published by Helmholz and Harrington [6] was so marked that the pictures are almost interchangeable. The mentality was subnormal. There was moderate hydrocephalus, the head being large and somewhat square, with moderately prominent frontal bosses. The bridge of the nose was broad and markedly depressed, and nasal breathing was considerably obstructed.



Dysostosis multiplex.

There was a profuse mucopurulent discharge in the nares. The palatal arch was high and narrow, with obstructive adenoids but small tonsils. Adenoidectomy failed to relieve the nasal obstruction, which was apparently due to the deformity.

The eyes were set markedly far apart and at first glance gave the impression that the corneas were abnormally large. A mongolian epicanthal fold was present. There was no exophthalmos, the palpebral fissure and the motility of the lids being normal. The ocular rotations were good in all directions. The sclera was not blue, or at least was less so than it frequently is in normal children. There was no injection of either the conjunctival or the ciliary vessels and no history of ocular inflammation. The corneas measured 11 mm. horizontally and presented a uniform ground glass haziness which appeared homogeneous under the corneal

6. Helmholz, H. F., and Harrington, E. R.: A Syndrome Characterized by Congenital Clouding of the Cornea and by Other Anomalies, Am. J. Dis. Child. **41**:793-800 (April) 1931.

microscope and was uniformly distributed throughout the parenchyma. The surface epithelium was smooth and intact. There were no tears in Descemet's membrane and no deposits thereon. The anterior chambers were of normal depth. The irides were normal in appearance and not tremulous. The pupils were moderately small, round and equal and contracted promptly and well to light stimulation. The reaction in accommodation was not recorded. After instillations of homatropine hydrobromide, cocaine hydrochloride and epinephrine hydrochloride there was only slight dilatation; with atropine sulfate a further, but still abnormal, enlargement was obtained. There was no increase in the corneal haze during mydriasis, nor was it decreased by pilocarpine nitrate. There was, however, a definite fluctuation in the density of the corneal cloudiness at subsequent observations. No synechiae were seen and the lens appeared clear. Examination of the fundus was made with the child under general anesthesia, and although the definition was somewhat impaired by the corneal condition the optic nerves were made out to be normal in color and outline and no gross pathologic condition was noted in the remainder of the eyegrounds. The ophthalmoscope revealed no marked refractive error. The baby was seen eight times, and at each visit the tension, observed by palpation (use of the tonometer being impossible), remained definitely within normal limits. The child followed lights and objects with alacrity and apparently saw well. The results of general physical, laboratory and roentgen examinations conformed to the usual descriptions of this disease.

The constant ocular manifestation of dysostosis multiplex is diffuse interstitial clouding of the cornea. It is sometimes the only ocular defect, but in 24 of the reported cases there were other abnormalities. Bindschendler, Rodier and Heintz-Bertsch [7] and Bindschendler [8] reported 2 cases in which the eyes were large and slightly prominent. No measurements were given. The intraocular tension was elevated at times. An anterior sclerotomy was done in 1 case and an Elliot trephining operation in the other. In neither instance did lowering of the tension have any effect on the corneal clouding. Davis and Currier [9] reported 2 cases (as instances of Morquio's disease) in which buphthalmos and optic atrophy were present. Jewesbury and Spence [10] reported 2 cases in which there were prominent eyeballs, but no corneal measurements were given. In Meyer and Okner's [11] case there was megalocornea, the corneas measuring 14 mm. in diameter, with normal tension. Berliner [4] reported "slightly larger than normal" corneas in 2

7. Bindschendler, L. L.; Rodier, J. M., and Heintz-Bertsch (Mme.): La polydystrophie du type Hurler, maladie congénitale et familiale, Rev. franç. de pédiat. **14**:116-134, 1938.

8. Bindschendler, J. J.: Polydystrophie du type Hurler chez au frère et une sœur, Bull. Soc. de pédiat. de Paris **36**:571-576 (July) 1938.

9. Davis, D. B., and Currier, F. P.: Morquio's Disease: Report of Two Cases, J. A. M. A. **102**:2173-2176 (June 30) 1934.

10. Jewesbury, R. C., and Spence, J. C.: Two Cases: (1) Oxycephaly and (2) Acrocephaly with Other Congenital Deformities, Proc. Roy. Soc. Med. (Sect. Dis. Child.) **14**:27 (April) 1921.

11. Meyer, S. J., and Okner, H. B.: Dysostosis Multiplex with Special Reference to Ocular Findings, Am. J. Ophth. **22**:713-722 (July) 1939.

of his cases but did not give any measurements. The tension was normal in both. In case 4 of Ellis, Shelton and Capon's [12] series there were 14 mm. corneas with normal tension. In the same case diffuse staining with fluorescein was observed. This is the only reported instance in which the anterior epithelium was not intact. These authors also reported 2 cases in which there were thick lids and 2 in which blepharitis was observed.

Subnormal dilatation of the pupils [13] after administration of mydriatics has been recorded in 4 instances, including the case reported here, and in 1 of these (Engel,[13b] case 2) there were sluggish pupillary reactions to light and in accommodation. Helmholz and Harrington [6] noted anisocoria in 1 case and narrowing of the palpebral fissures in another. The latter condition was also reported by Putnam and Pelkan.[14] These authors also reported strabismus, of which additional instances were present in Gasteiger and Liebenam's [15] case and case 2 of Hurler's original report.[1] Slot and Burgess [5] reported nystagmus and congested optic disks which, because of the color, were not well demarcated from the surrounding retina. Pale but probably not atrophic disks were noted by Engel.[13b]

The refractive error was measured in only a few instances, but hypermetropia of 7 D. and 9 D. was observed by Gasteiger and Liebenam [15] and myopia of — 8 D. by Berliner. An astoundingly short eyeball, of 37 D. (not 0.37 D.), was reported by Engel [13b] (case 2) as observed in a Chinese boy.

Because my own case was at first thought to be an instance of Morquio's disease and because some of the cases reported as cases of Morquio's disease are probably instances of dysostosis multiplex, it is pertinent to compare the two conditions.

Ten years ago Morquio,[16] of Montevideo, Uruguay, reported a distinct form of familial osseous dystrophy with dwarfism in some cases

12. Ellis, R. W. B.; Shelton, W., and Capon, N. B.: Gargoylism (Chondro-Osteo-Dystrophy, Corneal Opacities, Hepatosplenomegaly and Mental Deficiency), Quart. J. Med. **5**:119-140 (Jan.) 1936.

13. (*a*) Gillespie, J. B., and Siegling, J. A.: Dysostosis Multiplex, J. Bone & Joint Surg. **22**:171-175 (Jan.) 1940. (*b*) Engel, D.: Dysostosis Multiplex: Pfaunder-Hurler Syndrome (Gargoylism); Two Cases, Arch. Dis. Childhood **14**: 217-230, (Sept.) 1939.

14. Putnam, M. C., and Pelkan, F. K.: A Case of Scaphocephaly with Malformations of the Skeleton and Other Tissues, Am. J. Dis. Child. **29**:51-58 (Jan.) 1925.

15. Gasteiger, H., and Liebenam, L.: Beitrag zur Dysostosis multiplex unter besonderer Berücksichtigung des Augenbefundes, Klin. Monatsbl. f. Augenh. **99**: 433-447 (Oct.) 1937.

16. Morquio, L.: (*a*) Sur une forme de dystrophie osseuse familiale, Arch. de méd. d. enf. **32**:129-140 (March) 1929; (*b*) Sur une forme de dystrophie osseuse familiale, ibid. **38**:5-24 (Jan.) 1935.

of which pathologic ocular conditions are found. The general picture in these cases, in which the patients range in age from infancy to 35 years, is remarkably constant. The patients are short, with relatively long arms, and walk with a "duck waddle." The head is abnormally large, the bridge of the nose sunken and the neck so short that the head appears to rest on the trunk. There is a thoracic-lumbar kyphosis, with a narrow and prominent sternum. The lower extremities show flexed hips and loss of power but no muscular atrophy. Usually the intelligence is unimpaired. Roentgen examination shows a profound disturbance of epiphysial development, but except in a few cases the skull is normal. There is a familial factor, the disease being sex linked and following a definite hereditary pattern,[17] but healthy siblings are frequent. Consanguinity of the parents has been noted but is not usual. The affected child may be the first born or the youngest of several children. Ocular abnormalities occur but are not constant.

About 40 cases have been reported, which are reviewed here with respect to the ocular findings. There were 16 additional cases in five generations of the family described by Jacobson,[17] but he did not have the opportunity of observing them. Barnett,[18] Campbell [19] and Freeman [20] all remarked that the eyes were abnormally wide apart, and the last-named author added that his patient showed evidence of former mild chorioretinitis. Morquio [16a] reported blurred margins of the optic disks in 1 case (with normal vessels and normal vision), while in a second [16b] the eyegrounds were normal. Silverskiöld [21] remarked that 1 of his patients had exhibited myopia since childhood.

Giraud and Bert [22] reported the case of a patient who was first seen at the age of 34. The right eye was enucleated because of trophic and inflammatory changes which had appeared at 24 years of age. The side not operated on presented signs of irritation of the cervical portion of the sympathetic nervous system (exophthalmos with marked enlarge-

17. Jacobson, A. W.: Hereditary Osteochondrodystrophia Deformans (Morquio's Disease): Family with Twenty Members Affected in Five Generations, J. A. M. A. **113**:121-124 (July 8) 1939.

18. Barnett, E. J.: Morquio's Disease: Presentation of Two Cases, J. Pediat. **2**:651-656 (June) 1933.

19. Campbell, D.: Ueber eine typische Form des Zwergwuchses infolge gestörter enchondraler Ossifikation und die Frage ihrer verwandtschaft mit der Chondrodystrophie, Röntgenpraxis **3**:751-759 (Aug. 15) 1931.

20. Freeman, J.: Morquio's Disease, Am. J. Dis. Child. **55**:343-355 (Feb.) 1938.

21. Silverskiöld, N.: Atypical Achondroplasia and Its Peripheral Form, Acta radiol. **5**:223-236, 1926.

22. Giraud, G., and Bert, J. M.: La dystrophie osseuse de Morquio dans le carde des hyperlaxités familiales, Rev. neurol. **42**:845-856 (June) 1935.

ment of the palpebral cleft and mydriasis). The pupil reacted normally to light, but the reaction in accommodation appeared paralyzed. Despite any refractive error or change in the fundus, marked fatigue and lacrimation were experienced when close work was attempted. A second patient exhibited bilateral signs of irritation of the cervical part of the sympathetic nervous system: exophthalmos, enlarged palpebral cleft and mydriasis. The pupillary reactions were normal. Giraud attributed the symptoms referable to the sympathetic nervous system to lesions in the spine and the trophic disturbance in the first case to compression of the neurovascular pedicle of the eyeball.

Davis and Currier [9] reported the cases of 2 brothers, examined in the department of ophthalmology of the University of Michigan, with bilateral buphthalmos in which the impairment of vision was progressive and marked despite bilateral iridectomy. Four years later there was advanced atrophy of the optic nerves, with loss of the fine vessels. No suture lines were seen on roentgen examination of the skulls. The authors remarked that the shape of the heads was characteristic of scaphocephaly, with which blindness due to atrophy of the optic nerve is usually seen. Gillespie and Siegling,[13a] in a recent article, stated their belief that these cases were probably dysostosis multiplex.

Ruggles [23] reported 7 cases, in 2 of which the patients had cloudy corneas and were deaf and mentally defective. The disease became manifest at 2 or 3 months of age. It seems likely, as two groups of authors [24] have pointed out, that these were instances of dysostosis multiplex.

The remaining 14 reports [25] do not contain any references to the eyes.

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23. Ruggles, H. E.: Dwarfism Due to Disordered Epiphyseal Development, Am. J. Roentgenol. **25**:91-94 (Jan.) 1931.

24. Kressler and Aegerter.[3] Ellis, Shelton and Capon.[12]

25. Summerfeldt, P., and Brown, A.: Morquio's Disease, Arch. Dis. Childhood **11**:221-229 (Aug.) 1936. Brown, D. O.: Morquio's Disease, M. J. Australia **1**:598-599 (May 13) 1933. Coward, N. R., and Nemir, R. L.: Familial Osseous Dystrophy (Morquio's Disease), Am. J. Dis. Child. **46**:213-214 (July) 1933. Dale, T.: Unusual Forms of Familial Osteochondrodystrophy, Acta radiol. **12**:337-358, 1931. Brailsford, J. F.: The Radiology of Bones and Joints, ed. 2, London, J. & A. Churchill, Ltd., 1935. Grudzinski, Z.: Ueber eine neue mit Achondroplasie verwandte Krankheitsform, Fortschr. a. d. Geb. d. Röentgenstrahlen **38**:873-882 (Nov.) 1928. Sainz de los Terreros, C., and Lacalle, E.: Enfermedad de Perthes o de Morquio? Heredo-distrofia osteo-articular coxofemoral, Arch. españ. de pediat. **18**:412-423 (July) 1934. Meyer, F., and Brennemann, J.: A Rare Osseous Dystrophy (Morquio), Am. J. Dis. Child. **43**:123-135 (Jan.) 1932. Valentin, B.: Knochensystemerkrankung (atypische Chondrodystrophie, Osteochondropathia multiplex) und sogenannte Platyspondylia generalisata, Zentralbl. f. Chir. **57**:2038-2050 (Aug. 16) 1930. Warkany, J., and Mitchell, A. G.:

Dysostosis multiplex and Morquio's disease are both forms of dwarfism with which ocular defects occur. With both conditions there may be apparently large, prominent eyes and an abnormally great interpupillary distance. Points of differentiation are that in cases of Morquio's disease the condition is not manifest until the age of walking; there is a definite familial tendency; genu valgum is frequently present; the skull is normal, and the mentality is good. Ocular defects are infrequent. Dysostosis multiplex is recognized during the first year of life; there is no familial tendency; there are abnormalities of the skull; the cornea is invariably cloudy, and there are usually other ocular defects. The differentiation is of more than academic interest, as in cases of dysostosis multiplex the mentality is almost always impaired.

SUMMARY AND CONCLUSIONS

Dysostosis multiplex and Morquio's disease are forms of dwarfism with which ocular abnormalities occur. Cloudy cornea is a constant feature of dysostosis multiplex. Other ocular defects are fairly frequent and are reviewed.

Cloudy cornea is not a feature of Morquio's disease. Ocular defects are not frequent. Those reported are enumerated.

Some of the cases reported as instances of Morquio's disease were probably cases of dysostosis multiplex.

1569 Paulsen Medical and Dental Building.

Atypical Chondrodystrophy, J. Pediat. **4**:734-745 (June) 1934. Gamboa, M., and Salvatti, A. A.: Osteocondrosis generalizada enfermedad de Morquio, Bol. y trab. de la Soc. de cir. de Buenos Aires **22**:280-297 (June 15) 1938. Pohl, J. F.: Chondro-Osteodystrophy (Morquio's Disease) Progressive Kyphosis from Congenital Wedge-Shaped Vertebrae, J. Bone & Joint Surg. **21**:187-192 (Jan.) 1939. Marottoli, O. R., and de Azecuénaga, S.: Osteocondrodistrofia generalizada enfermedad de Morquio, Rev. méd. de Rosario **28**:1265-1274 (Dec.) 1938; An. de cir. **4**:387-395 (Dec.) 1938. Jacobson.[17]

MECHANICS OF OPTIC NERVE TRACTION ON THE RETINA DURING OCULAR ROTATION

WITH SPECIAL REFERENCE TO RETINAL DETACHMENT

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In attempting to study the possible relation between retinal detachments and traction stress of the optic nerve on the retina during ocular rotation, I have approached the problem from the standpoint of applied mechanics. When the eye is rotated to a considerable degree in any direction the optic nerve has a tendency to exert traction on the retina in the region of the disk. There is not enough slack in the orbital portion of the nerve to prevent this pull in the extreme horizontal positions. The traction is dependent on the anatomic position of the globe, the degree of rotation and the length of the nerve. Such traction can be made manifest entoptically. Von Helmholtz, Purkinje and Czermak have described a circular spot of "light" which is evoked in the region of the blindspot by the lateral movements of the eyes. This spot, in a modified form, may be observed with the eyes open and directed in extreme horizontal rotation on an illuminated white surface. The dark scotoma of the nerve head is seen surrounded by a brilliant border. If adduction and abduction are practiced in a completely darkened room one will observe circles of pale blue around the locus of the blindspot. In the nasally deviating eye a clearer image is induced. The size of the circle is considerably larger than the expected size of the blindspot, and one easily realizes that these luminous areas are the result of traction from the optic disk extending to the adjacent retinal elements. The circles are solidly blue, except for a vague defect which corresponds to the blindspot.

I have noticed another interesting manifestation which occurs when the lateral movements are performed rapidly in the dark. Several vivid arcs of blue "light" cross the lower quarter of the luminescent spot; these are more brilliant than the pale blue of the circle. The concavity of the arcs is directed upward. The nasally moving eye provides the more brilliant striations.

It is thus subjectively apparent that extreme rotation of the eye induces traction on the portion of the retina surrounding the nerve head and that nasal rotation is a more potent stimulus than temporal rotation.

Where in the ocular swing does the traction begin to be exerted, and why is the traction more evident in adduction? How far from the disk

is the traction exerted? To answer these and other related questions I have tried to illustrate the mechanics involved by representations of the globe, the optic nerve and the orbit, drawn to scale. I have checked these with drawings magnified to four times natural size. The following anatomic data, which constitute the basic measurements used in the sketches, are average figures:

Length of the medial wall of the orbit	40.9 mm.[1]
Length of the lateral wall of the orbit	46.2 mm.[1]
Angle between the medial and lateral walls of the orbit	45 degrees
Distance from the cornea anteriorly to the most recessed point of the lateral wall	16.5 mm.[2]
Distance from the center of the pupil to the center of the orbital opening (the pupil is lateral to the center of the orbital opening)	1.5 mm.
Distance from the anatomic center of the globe posteriorly to the center of rotation	2.0 mm.
Distance from the macula to the insertion of the lateral margin of the optic nerve	3.0 mm.
Distance from the center of the optic nerve downward to the level of the macula	1.0 mm.[3]
Limit of adduction	50 degrees
Limit of abduction	50 degrees
Limit of downward rotation	50 degrees
Limit of upward rotation	45 degrees

In spite of the fact that the ophthalmoscope reveals that the disk is higher than the macula and that the projected position of the blindspot is lower than the fixation point, nearly all anatomic descriptions erroneously place the disk 1 mm. lower than the macula.

The medial orbital wall is practically in the anteroposterior line.

The optic nerve is fixed to the roof of the optic canal by adhesions between the nerve coverings and the contiguous periosteum of the optic canal.

The average lengths for the optic nerve stated in textbooks are so variable that I have kept the length of the nerve as an unknown quantity in this study and have determined its value from the drawings.

In these measurements I concerned myself only with the orbital portion of the nerve, since the nerve is anchored to the periosteum of the optic canal. The optic foramen is therefore a stationary pivot around which the orbital segment of the nerve swings.

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1. Emmert, cited by Norris, W. F., and Oliver, C. A.: System of Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1897, vol. 1, p. 116.

2. Jackson, cited by Fuchs, E.: Textbook of Ophthalmology, translated by A. Duane, ed. 8, revised, Philadelphia, J. B. Lippincott Company, 1924, p. 55.

3. Testut, L.: Traité d'anatomie humaine, ed. 8, Paris, Gaston Doin, 1930, vol. 3, p. 61.

From the drawings of the eye in its primary position I have determined the following measurements, which are of interest:

Linear distance between the centers of the disk and the optic foramen	29 mm.
Angle between the medial wall of the orbit and the general line of the optic nerve	28 degrees

A glance at charts 2 and 3 will show that when the eye is turned temporally the retina is compressed toward the disk from the temporal side and pulled away from the disk on the nasal side; when the eye is turned nasally the retina is compressed toward the disk from the nasal side and pulled away from the disk on the temporal side.

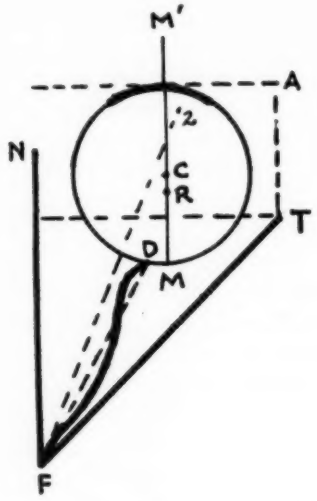

Chart 1.—In this and in accompanying figures *NF* indicates the nasal wall of the orbit; *TF*, the temporal wall of the orbit; *F*, the foramen; *AT*, the distance of the cornea anterior to the temporal orbital wall; *DF*, the optic nerve; *MM'* the visual axis; *F2*, the line bisecting the angle of the orbit; *C*, the anatomic center of the globe, and *R*, the center of rotation.

The optic canal is known to make an angle of 37 degrees with the medial wall of the orbit. When the eye is in extreme nasal rotation the nerve makes an angle of 41 degrees with the medial wall; that is, it bends toward the temple at the foramen to make an angle of 4 degrees with the optic canal. When the eye is in extreme temporal rotation the nerve makes an angle of 13 degrees with the medial orbital wall; that is, it bends toward the nose at the foramen to form an angle of 24 degrees with the optic canal. The angulation in this position may be enough to strangulate the pulsation in the retinal veins.[4]

4. Graves, B.: Brit. J. Ophth. **6**:237, 1922.

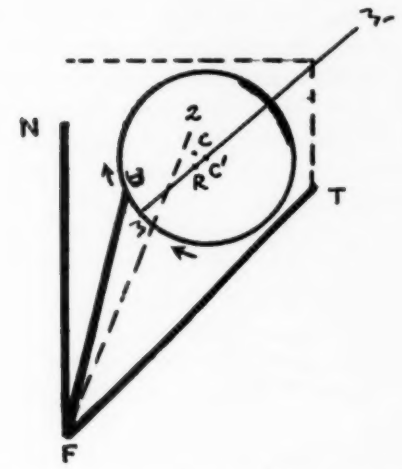

Chart 2.—*C'* indicates the new position of *C* when the eye is turned 50 degrees temporally. The arrows show how the retina is compressed toward the disk from the temporal side and pulled away from the disk on the nasal side.

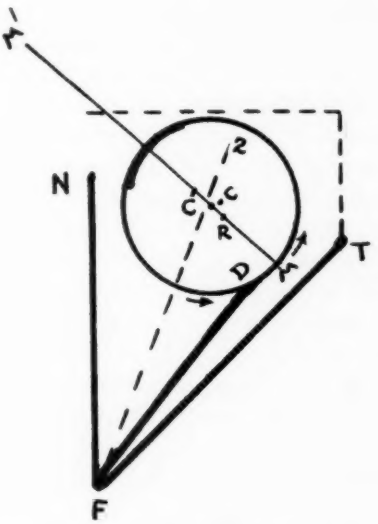

Chart 3.—*C'* indicates the new position of *C* when the eye is turned 50 degrees nasally. The arrows show how the retina is compressed against the disk from the nasal side and pulled away from the disk on the temporal side.

When the eye is turned 50 degrees nasally the following changes occur: The macula lies 4.0 mm. behind the temporal edge of the orbit. The lateral border of the nerve moves to a calculated position 35.25 mm. from the foramen; this measurement is made from the new position of the macula. A slight stretching of the portion of the retina adjacent to the lateral border of the disk occurs, so that the border of the disk does not move quite the full 35.25 mm. The temporal border of the disk lies 6.0 mm. behind the temporal edge of the orbit. The globe recedes nearly 1 mm. and shifts about 1.5 mm. nasally.

When the eye is turned 50 degrees temporally the following changes are noted: The medial border of the nerve, judging from the new position of the macula, moves to a calculated position 34.75 mm. distant from the foramen. A slight stretching of the portion of the retina adjacent to the medial border of the disk occurs, so that the border of the disk does not move quite the full 34.75 mm. The globe recedes nearly 1 mm. and shifts temporally about 1.5 mm.

When the eye is turned upward 45 degrees the lower margin of the disk is 33.4 mm. from the foramen.

When the eye is turned downward 50 degrees the upper margin of the disk is 34.25 mm. from the foramen.

It is evident that the optic nerve and the temporally adjacent retinal tissues must be under stretch to the extent of 35.25 mm. when the eye is in extreme medial rotation. This is the maximum physiologic extension to which the nerve and the retina are subjected in the idealized eye.

The lengths of the orbital part of the optic nerve which are mentioned in textbooks depend chiefly on measurements made by Weiss [5] in 1890. Weiss based his values on 60 dissections and ascribed to the nerve an average length of 24 mm., with a slack of 5.6 mm. Schaeffer's [6] figures are slightly higher, the average length being 27.5 mm. Wexler [7] gives the length as 25 to 35 mm.; presumably the average would be 30 mm.

The traction circles are observed when the eye is within about 5 degrees of its abduction limits and slightly sooner on adduction. When traction begins, according to this estimate, the temporal and nasal borders of the disk are both 34 mm. distant from the foramen. This distance was determined on a drawing of the eye properly rotated; the new position of the macula was used as a point relative to which the temporal edge of the disk was plotted. Since the ultimate calculated position of the margin of the disk is 35.25 mm. from the foramen on

5. Weiss, L.: Beiträge zur Anatomie der Orbita, Tübingen, H. Laupp, 1890, pt. 2.

6. Schaeffer, J. P.: Am. J. Ophth. **4**:451, 1921.

7. Wexler, D., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 66.

nasal rotation, the optic nerve and the temporally adjacent retinal tissues must together undergo a stretching of 1.25 mm. in that position.

When traction starts on abduction, the nasal border of the disk is 34 mm. from the foramen; its calculated ultimate position will be 34.75 mm. from the foramen; therefore, the nerve and the nasally adjacent retinal tissues must be stretched 0.75 mm. in extreme abduction.

If tension on the nerves begins at a distance of 34 mm. from the foramen, this must be the length of the nerve. In its most distorted position, as has been stated, the nerve and the adjacent tissues must be displaced 35.25 mm. from the foramen; therefore, the nerve and the temporally adjacent retinal tissues must together be capable of a maximum elastic displacement of 1.25 mm. In spite of its fixation to the roof of the optic canal, the optic nerve must stretch to some extent by virtue of oblique traction transmitted through its fascicular structure; most of its fibers are free from direct attachment to the canal and can slide against each other a slight distance in opposite directions. The effect is like pulling on the opposite corners of a rectangle, thereby increasing the length of the connecting diagonal. In addition, one might expect the sclera to yield somewhat by a slight flattening of its curvature. The same traction is simultaneously transmitted to the retina. The retinal membrane is ductile to some extent and by stretching quickly absorbs its share of the traction before it extends much beyond the disk. The elasticity of the normal retina is attested by the numerous instances of rupture of the choroid without rupture of the overlying and adherent retina.

In extreme upward rotation the nerve and the inferiorly adjacent retinal tissues must undergo no stretching at all; indeed, there is a slack of 0.6 mm. of nerve. I have been unable to see the traction circles with the eye in this position. When the eye is in extreme downward rotation the nerve and the superiorly adjacent retina must undergo a stretching of only 0.25 mm. Downward rotation produces only the feeblest entoptic traction circles; I believe that movement in the vertical plane may be ignored in the consideration of retinal traction by the optic nerve.

The traction produced in positions intermediate between the vertical and the horizontal plane is about equal to the average of the tractions corresponding to the nearest vertical and horizontal limits.

According to my drawings, since the distance separating the disk and the foramen in the primary position is 29 mm. and the length of the nerve is 34 mm., there must be a slack of 5 mm. when the eye is directed forward.

The length of 34 mm. which my drawings indicate for an average nerve is 10 mm. greater than the average of Weiss and 4 mm. greater than the average of Wexler. In explaining these differences some

allowance should be made in anatomic measurements for normal shrinkage of the tissues after death and for shrinkage from preservatives. It must furthermore be borne in mind that my drawings are based on average sizes and positions of the globe and the orbit, a fortuitous combination of norms rarely encountered in a single subject. My draw-

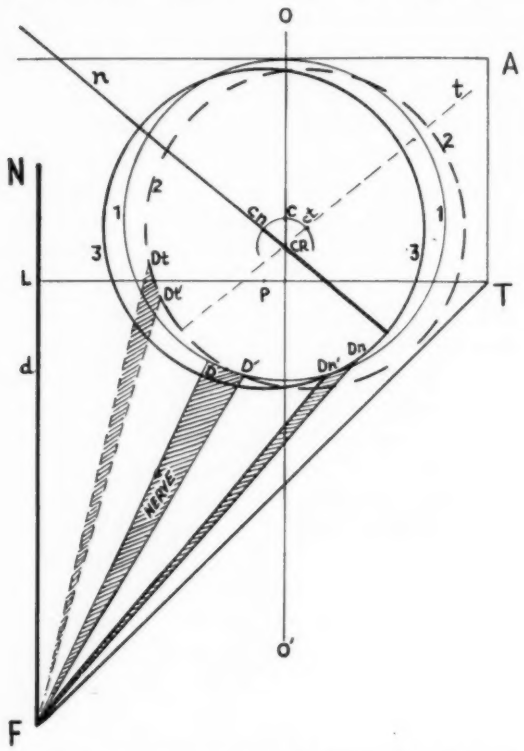

Chart 4.—Illustration of the limits of ocular rotation in the horizontal plane. *CR* indicates the center of rotation. If an angle of 50 degrees is laid off nasally and temporally with *CR* as the center, the path of *C* will be along the arcs *Ccn* and *Cct*, respectively. The visual axis *n* will pass through *cn* and *CR* when directed 50 degrees nasally, and from the macular position the new position of the nerve is readily determined. When the eye is turned 50 degrees temporally the visual axis *t* will pass through *ct* and *CR* and thus fix the location of the macula, from which the new position of the nerve is again plotted.

L indicates a projection of *T* against the nasal orbital wall. *P* is the center of *TL*. *OO'* passes through a point 1.5 mm. temporal to *P* on *LT* in an anteroposterior direction, and in this plane lies the center of the pupil. The limits of the disk are *Dn* and *Dn'* when the eye is turned nasally, *Dt* and *Dt'* when the eye is turned temporally and *D* and *D'* when the eye is in the primary position. The length of *DD'* is laid off as 3 mm., the diameter of the optic nerve at its insertion into the sclera, because the traction force is applied to the globe at this union. Circle 1 indicates the primary position of the globe, circle 2 the position on extreme temporal rotation and circle 3 the position on extreme nasal rotation.

ings must be revised when more is known concerning the vagaries of the center of rotation. It is not stationary as my sketches indicate but is itself a variable eccentric point. No data are available at present for the locus of the center of rotation beyond 35 degrees of ocular rotation. My measurements for the nerve would have to be altered if this were known, but the amendments would not be considerable.

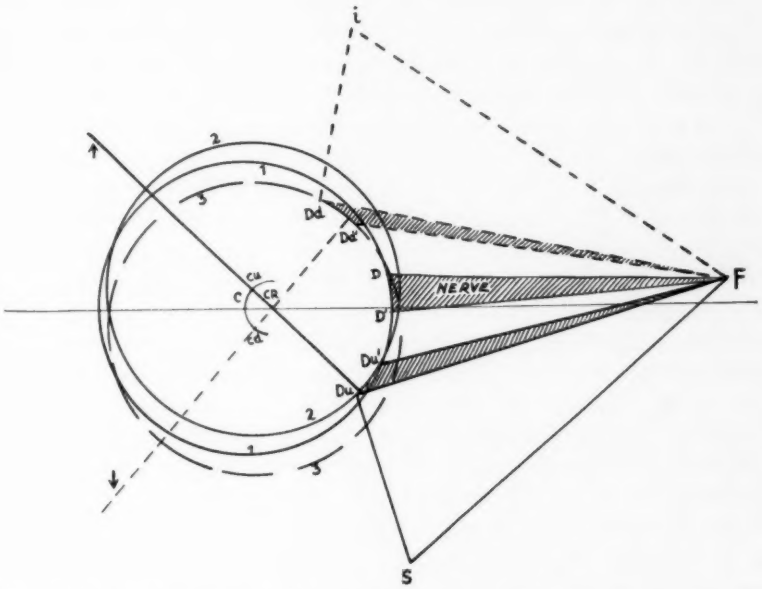

Chart 5.—Lateral projection of chart 4, illustrating limits of ocular motility in the vertical plane. The disk moves vertically along an arc whose radius is 11.25 mm. (By referring to chart 4 it will be seen that the movement up and down of *DD'* will be through a plane passing through the midpoint of *DD'* and parallel to *OO'*. The tropic through the circle of the eye at the midpoint of *DD'* parallel to *OO'* will necessarily be smaller than the equator, which lies in the plane *OO'*, and will have a radius of 11.25 mm. The circles in chart 5 consequently have a radius of 11.25 mm.) When the eye is raised upward 45 degrees *C* travels to *cu*. When the eye is lowered 50 degrees *C* travels to *cd*. The limits of the disk are *Du* and *Du'* when the eye is raised, *Dd* and *Dd'* when the eye is lowered and *D* and *D'* when the eye is in the primary position. *FS* indicates the full length of *F Du*. *Du S* takes into account the temporal deviation of the nerve while it is in this position and is represented in chart 4 by the distance between *DD'* and *d*. *Fi* is the full length of *FDd*. *Ddi* allows for the temporal deviation of the nerve in this position. Circle 1 indicates the primary position of the globe, circle 2 the position on extreme upward rotation and circle 3 the position on extreme downward rotation. The true length of a line which forms an angle in space with three reference planes will be the hypotenuse of a right triangle of which the two sides are the rectangular projection of the line on one of the reference planes and its projection on the intersection of the other two planes.

If the eye revolved around its anatomic center, extreme abduction would cause a stretching of the nerve and the retina of 1.25 mm., while extreme adduction would demand a stretching of 2.75 mm.

If the nerve were only 30 mm. in length (Wexler), almost no ocular rotation would be permitted, since there would be only 1.0 mm. of slack. This would allow only 5 degrees of rotation nasally and about 15 degrees temporally. If the nerve were 24 mm. in length (Weiss), the eye, allowing for the slack in the nerve, would have to lie so far back toward the apex of the orbit that there would be insufficient orbital space at that level to accommodate it and the cornea would project only 7.75 mm. anterior to the temporal margin of the orbit.

As nearly as I can determine from entoptic observations, the effects of traction extend for about 1 mm. around the disk in the normal eye. This estimate is made by judging the size of the traction circle and comparing it with the size of the entoptic blindspot. One would expect to find an enlargement of the blindspot on extreme temporal rotation to correspond with the traction circle. I must confess that the tangent screen fails to reveal this convincingly in the normal eye; the difficulty of maintaining unfaltering fixation is exceedingly great. In cases of pronounced myopia, in which traction would be more marked, the inevitable peripapillary atrophy produces an enlargement of the blindspot which masks whatever enlargement might be caused by traction. Plotting of the blindspot is not feasible in extreme adduction, where traction effects would be most evident, because the nose interferes. Certainly the evidences of traction do not extend over the 3 mm. distance to the macular area. This may be ascertained by observing entoptically that the terminals of the vertical vessels in the macular region do not alter their slope in extreme horizontal rotation. An additional proof of the undisturbed state of the macula under these conditions lies in the fact that when the macula fixates on a straight vertical line while the eye is in extreme abduction, the continuity of the line is preserved without distortion in the fixation area.

I mention these points in order not to overemphasize the role of optic nerve traction as a force in producing retinal detachments; doubtless it is not immediately responsible in all cases. If it did invariably assume major importance, one would expect to find the incidence of detachment proportionate to the degree of myopia, because in cases of extreme myopia the retina is stretched to an excessive degree and the retinal elasticity is at a minimum. The exophthalmos in cases of pronounced myopia would further augment the effects of traction by taking up the slack in the nerve. The greatest number of detachments, however, occur in cases of moderate, not extreme, myopia. (It is possible that patients with exophthalmos avoid extreme duction strains on the eye by moving the head.)

One feels instinctively that the ocular movements do have a tendency to aggravate an existing detachment, to imperil the restoration of the retina when the injury has been repaired and even to inaugurate a separation. Two possible explanations of the modus operandi of retinal detachment other than optic nerve traction come to mind.

The first relates to the phenomenon of inertia. A rapid motion of the eye will cause the semifluid vitreous to lag momentarily behind the retina. It soon keeps pace with the retina, but when the eye has stopped its swing the vitreous will continue its motion. Adhesions between the retina and the vitreous—normal, pathologic or postoperative—will tend to pull the retina from its bed at the beginning and at the end of the ocular swing.

A second cause which has been suggested is the pressure by the extraocular muscles exerted on the globe and transmitted to the retina. The frequency of tears in the region underlying the superior oblique muscles is provocative of such a thought; and the idea may be correlated with the constant exertions of the superior oblique muscles in the acts of reading and close work.

Nevertheless optic nerve traction is a force which cannot be wholly ignored. Witness the prevalence of myopic peripapillary conus and its frequently progressive nature.[8] Such clinical findings may be explained by the drawing away of the choroid during the ocular movements or by traction atrophy. This retraction is possible because the choroid, unlike the retina, is not directly adherent to the nerve. The movements toward the nose, as has been said, produce the greatest tension, and this is corroborated clinically by the great prevalence in cases of myopia of temporal conus; it is the temporal portion of the retina and choroid which are under stress in this position. If a retina is already fated by a pathologic background for detachment, a slight pull by the optic nerve may well be the trauma precipitating detachment, and little pull would be required. Rotation nasally would be particularly hazardous. In a case of axial myopia of 10 D. the retina would be under a maximum stress equivalent to over 3 mm. of nerve-retina displacement, as compared with the 1.25 mm. of stress in the normal eye. It is significant that the great preponderance of tears are in the temporal part of the retina in cases of detachment; I believe it reasonable to incriminate traction transmitted temporally from the disk as the responsible agent in many of these instances, remembering always that such eyes are not normal in their resistance to traction. The projected location of the bright striae which I have described in the lower portion of the entoptic traction circle indicates that the portion of the retina adjacent to the upper border

8. Friedenwald, J. S.: Pathology of the Eye, New York, The Macmillan Company, 1929, p. 251.

of the disk is under particular tension during horizontal rotation. This is undoubtedly due to the adhesions between the roof of the optic canal and the upper nerve fibers; they prevent "give" in the upper fibers, and the traction is transmitted most forcibly to that part of the retina which lies in connection with these fixed fibers, namely the upper temporal quadrant. It is again significant that most of the temporal tears are concentrated in the upper temporal quadrant.

I believe it likely that all three of the factors which I have mentioned act on the retina during ocular rotation and that the relative importance of each may vary in different cases. From any point of view, the practice of preventing ocular movement by means of stenopeic goggles is based on sound reasoning.

Chart 6.—Schematic distribution of 75 retinal tears (after Gonin). *T* indicates temporal side and *N* nasal side. The outer circle indicates the ora serrata and the other circles the retinal zones situated at a distance of 2, 4, 6, 8 and 10 papillary diameters from the ora serrata. The symbol V indicates a tear; the dots, holes (perforations without any tearing), and the lines, disinsertions of the retina.

SUMMARY

The question of optic nerve traction on the retina at the extreme limits of ocular rotation has been treated as a problem in descriptive geometry; idealized and averaged data were used.

The calculated length of the optic nerve is 34 mm., with 5.0 mm. of slack in the primary position.

In extreme abduction the nerve and the portion of the retina nasally adjacent to it must together stretch 0.75 mm.

In extreme adduction the nerve and the portion of the retina temporally adjacent to it must together stretch 1.25 mm.

These figures are subject to revision in the light of more data concerning the center of rotation.

Entoptic evidence is presented that the portion of the retina adjacent to the upper border of the disk is under particular stress during these lateral movements.

These observations have been correlated with the predominant locations of tears in cases of retinal detachment, and an attempt has been made to establish a clinical relation.

NOTE.—For the sake of simplicity I have taken the liberty of making the visual and the optic axes coincident in the drawings. I have also used the outside rather than the inside measurements of the globe and nerve since the forces exerted on them are applied externally.

CHARACTERISTICS OF SENSITOMETRIC REFRACTION

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AND
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In a recent publication [1] a new subjective method of refraction was described and clinical data obtained by the procedures were presented. This method involves the determination of the refractive power which will produce maximal visibility [2] as appraised by thresholds of brightness contrast rather than of visual acuity. By providing a convergence target of sufficient size and indistinctness, the method permits the functioning of convergence in the proved absence of any adequate stimulus for either positive or negative relative accommodation. Furthermore, in contrast to the usual methods of refraction, the procedures are identical in the static and dynamic tests. Hence the specific characteristics of the accommodation-convergence coupling are clearly revealed. For example, it has been shown that normal adult nonpresbyopic eyes in a so-called physiologic state of rest are accommodated for a point anterior to the retina and that there is no evidence of a lag of accommodation at the usual reading distance, such as has been postulated.

In the present paper the relations between (1) visibility and refraction and (2) accommodation and convergence are presented in detail for a typical adult emmetropic subject. These precisely delineated relations not only indicate the relative extent to which vision is blurred by refractive errors of various magnitudes but reveal the progressive changes in the refractive state of the eyes concomitant with binocular convergence at various fixational distances between infinity and the near point. These data are particularly significant because all determinations of the refractive states of the eyes are based on the absolute and fundamental criterion of maximal visibility rather than on some arbitrary standard of "normal" visual acuity. Furthermore, a high degree of precision is obtained by

From the Lighting Research Laboratory, General Electric Company.

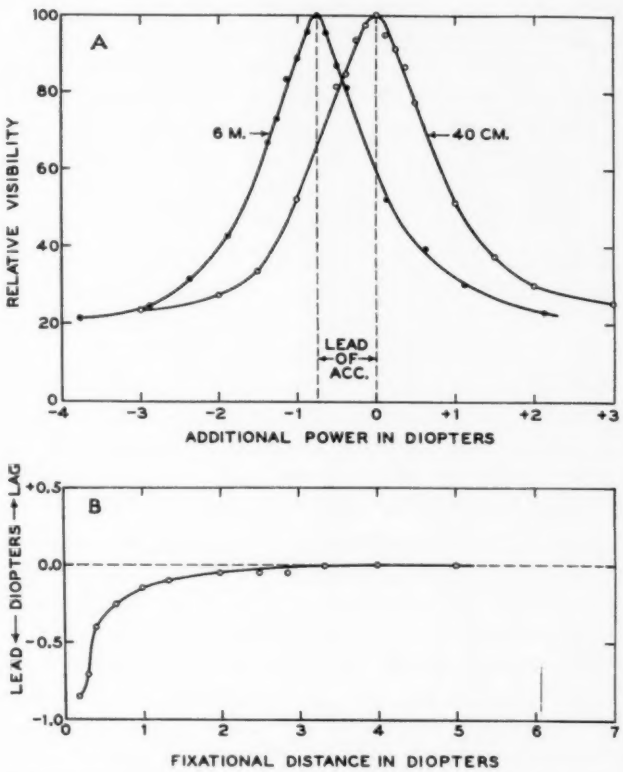
1. Luckiesh, M., and Moss, F. K.: New Method of Subjective Refraction Involving Identical Technics in Static and Dynamic Tests, Arch. Ophth. **23**:941 (May) 1940.

2. Luckiesh, M., and Moss, F. K.: Supra-Threshold Visibility, J. Optic. Soc. America **30**:62, 1940.

repeating under identical physical conditions the quantitative measurements of visibility, so that each measurement quantitatively influences the final result.

VISIBILITY AND REFRACTION

The curves in *A* of the figure show the relations between relative visibility and refraction for fixational distances of 6 meters and of 40 cm.,



Data for typical adult emmetropic subject. *A*, relations between visibility and refraction for fixational distances of 6 meters and 40 cm. The two curves are plotted from the average results obtained on 8 different occasions, and to facilitate comparison the visibility data are plotted on a percentage basis. The actual values for maximal visibility as read from the scale of the sensitometer were not the same for the two distances, since such factors as the illumination of the test object and its angular size differed. The refractive powers corresponding to maximal visibility in the 8 tests are as follows:

At 6 meters.... — 0.80, — 0.80, — 0.70, — 0.85, — 0.70, —0.70, —0.70, — 0.80
Average, — 0.75

At 40 cm..... 0, 0, + 0.10, 0, — 0.05, — 0.05, 0, 0
Average: 0

B, relation between accommodation and convergence, as determined from 2 concordant examinations. Data are for the right eye, with accurately controlled convergence.

respectively, as determined from eight concordant examinations made on different occasions. In all tests the subject maintained binocular fixation during the monocular examinations of the refractive states of the right eye. It will be noted (1) that the visibility-refraction curves obtained under so-called static and dynamic conditions are practically identical in shape, (2) that the curves are symmetric about their respective axes of maximal visibility and (3) that the two curves can be made practically congruent by laterally translating one of them 0.75 diopter. The dioptric displacement between the two curves is to be described as a lead of accommodation at the far point, since the refractive state of the eye is such that additional negative power is required to produce maximal visibility at distance. Furthermore, such a lead of accommodation is characteristic of all adult emmetropic subjects so far examined. It will also be observed from *A* of the figure that the lead of accommodation remains reasonably constant while the test object is reduced from maximal visibility to virtual invisibility by either convex or concave lenses.

The symmetry of the curves relating visibility and refraction indicates that relative accommodation is avoided in the sensitometric procedures at these fixational distances, except perhaps to a small extent for extreme degrees of artificial ametropia. However, any such slight distortion is unimportant, since the extremities of the visibility-refraction curve would not be used in clinical determinations of refractive errors. At the same time, a consideration of the data obtained under extreme dioptric conditions emphasizes the completeness of the control of accommodative functions in the sensitometric procedures. For example, the subject was able to obtain reliable and reproducible measurements of visibility even with lenses of -3 and $+5$ diopters, respectively, before the right and left eyes. For this subject, the $+5$ diopter lens before the left eye served to obliterate the image of the test object in this eye and thus to confine the refraction to the right eye. However, the subject was not aware that any refractive difference existed between the two eyes, nor did he experience the slightest degree of accommodative fatigue as a result of an actual difference of as much as 8 diopters in the course of many measurements. Obviously, a refractive differential of this order of magnitude would have been intolerable had the procedures permitted the functioning of relative accommodation to any appreciable degree. It should also be stated that similar responses and reactions were obtained with all the other subjects examined.

It will be noted from *A* of the figure that the relation between visibility and refraction, from 0.25 to 1 diopter on either side of the point of maximal visibility, may be represented approximately by straight lines. This characteristic, which is attributable to the particular scale of visibility associated with the gradient filters of the sensitometer,[2] obviously facilitates the application of the sensitometric method in clinical

practice.[1] In the region of maximal visibility the character of the curves is influenced by such factors as aberrations and depth of focus of the eye. Since the curves for different subjects vary in the sharpness of the "apex" and in the slope of the "straight line" portions, these characteristics provide additional information on the visual mechanisms of the individual subject. As the refractive error is increased the visibility of the test object asymptotically approaches a limit which corresponds to the limit in contrast sensitivity of the subject. It is of interest to note also that the decrement in visibility is somewhat greater for the addition of an extreme amount of negative power than it is for the addition of the same amount of positive power. This result is in accord with optical principles, and the fact that it holds good for both distances is an indication of the inherent precision of the sensitometric method.

It is evident from these relations that relative accommodation, either positive or negative, would be required to obtain maximal visibility at both long and short range. If the indicated correction for distance were supplied, maximal visibility at distance would be obtained without the exercise of relative accommodation. Incidentally, such a correction has been found suitable for operators of motion picture projectors, whose visual tasks involve critical seeing at distance for prolonged periods. For such subjects the advantage of the correction for distance is probably due as much to increased sharpness in vision as to greater ease in seeing. This viewpoint is suggested by the fact that higher degrees of visibility are usually obtained when the lead of accommodation is offset by concave lenses than when the compensation is made by negative relative accommodation. However, a complete far point correction would require excessive compensation by means of positive relative accommodation for tasks at close range. Since this form of compensation involves parasympathetic activity, it would be associated with subjective fatigue. On the other hand, the near point correction would yield maximal visibility at close range without the exercise of relative accommodation, while at least approximate maximal visibility at distance would be obtained through a compensation of the lead of accommodation by negative relative accommodation. Since the latter involves sympathetic functions, it would not ordinarily be associated with subjective fatigue.

ACCOMMODATION AND CONVERGENCE

The refractive state of a typical normal adult eye, with binocular fixation and in the absence of optical stimuli for relative accommodation, is shown in *B* of the figure for various fixational distances between 6 meters and 20 cm. These data reveal that the lead of accommodation decreases rather abruptly, but regularly, as the fixational distance is decreased from 6 meters to about 1 meter and that for shorter ranges the eye is practically emmetropic. However, a progressively decreasing

lead and then an increasing lag of accommodation have been observed with some so-called emmetropic subjects for the range of fixational distances shown in *B* of the figure. The gradual reduction of the lead of accommodation as the fixational distance is decreased is to be considered direct evidence that the lead of accommodation is not an artefact arising from systematic errors in the sensitometric method. This conclusion is also supported by the fact that the observed lead of accommodation varies markedly among a group of so-called emmetropic subjects.

It appears that at a disance of 6 meters the refractive power of the eyes of the typical subject under consideration exceeds that required by about 0.75 diopter; that is, an addition of this amount of negative power is required to obtain maximal visibility. At a distance of 1 meter the excess in plus power amounts to only 0.166 diopter, as will be noted from *B* of the figure. Since an additional 0.83 diopter is required when the point of fixation is shifted from 6 meters to 1 meter and since 0.75 diopter is available because of the lead of accommodation, it appears that the excess of 0.166 diopter at a distance of 1 meter is due to convergence accommodation amounting to 0.25 diopter. Thus, only a fraction of the total amount of additional plus power required for such a shift in fixation is supplied as a result of increased convergence. However, it will be noted that some excess power is supplied notwithstanding the fact that it blurs the vision. At a fixational distance of 0.5 meter, an increase of 1.83 diopters is needed, of which 0.75 diopter is available because of the lead of accommodation. Since the eye is practically emmetropic at this distance, it may be assumed a priori that the deficiency of 1.08 diopters is supplied by convergence accommodation. It appears that for distances between 0.5 and 0.2 meter all of the additional power required by a given change in fixation is derived from the accommodation-convergence coupling. For the latter range of fixational distances the relation between these two functions corresponds to that usually considered as normal.

This analysis indicates that the relation between accommodation and convergence for fixational distance between infinity and the near point is not one of direct proportionality, such as is customarily assumed. Rather it appears that the amount of convergence accommodation induced by a given dioptric shift in fixation increases from a practically negligible proportion of that required to the total amount required as the fixational distance decreases.

It also appears from evidence so far obtained that the refractive errors indicated by dynamic sensitometry are in the same direction as and are similar in magnitude to those indicated by usual methods after the induction of cycloplegia and by the "fogging" method of refraction after the adoption of various expedients in the latter procedures. Although the ophthalmic corrections indicated by the usual static exami-

nation approximate those indicated by dynamic sensitometry, the sensitometric data are more readily interpretable because they are obtained with binocular convergence and under conditions of normal nervous control of the ocular musculature. Obviously, these advantages are important when abnormal relations between accommodation and convergence are involved, as well as when precise corrections are demanded for near vision. Thus, the sensitometric method adds to the science of refraction a subjective test which is at least as significant and precise at all distances as are present static tests either under conditions of normal muscular control or after the induction of cycloplegia. Furthermore, sensitometric examinations at critical distances provide data for determining the direction and the extent of the relative accommodation which will be required with any prescribed correction at any distance.

PRINCIPLES OF SUŚRUTIAN OCULAR THERAPY INTERPRETED IN THE LIGHT OF MODERN OPHTHALMIC SCIENCE

WITH A BRIEF STUDY ON THE EVOLUTION OF OCULAR THERAPEUTICS FROM THE PREHISTORIC AGE UP TO THE MODERN PERIOD

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In this paper an attempt has been made to enumerate briefly the essential principles of Suśrutian ocular therapy and to interpret and evaluate them in the light of modern ophthalmic science. An attempt has also been made to trace, as far as the scope of the paper allows, the evolution of ocular therapeutics from the prehistoric age to the present time.

Ever since the prehistoric age when Suśruta [1] systematically built ophthalmic surgery on a scientific basis, the medical world has witnessed manifold changes in the domain of ophthalmology. Thanks to the pains-taking and learned researches of the world scientists of succeeding generations, numerous innovations in technic have been introduced and many therapeutic advances made. Paradoxic though it may appear, notwithstanding the investigations carried out from time to time by the medieval as well as the modern ophthalmologists, the essential principles of ocular therapy as taught and practiced by Suśruta have remained fundamentally unchanged. It may be mentioned, for example, that many of the teachings of Suśruta on ocular therapy have been followed by Hippocrates [2] as well as other eminent medical authorities of the post-Suśrutian period, Celsus,[3] Albucasis,[4] Aeginata,[5] Rhazes,[6] Vagbhatta,[7] Chakrapani Datta,[8] Sārangadhara [9] and others.

From the Suśruta Clinic.

1. Suśruta Saṁhitā, with Dallana Acharya's Commentary, edited by Yādavji Sharma, ed. 2, Bombay, Nirnaya-Sagara Press, 1931, Uttara Tantra, chaps. 1-19.

2. Hippocrates: The Genuine Works of Hippocrates, translated by F. Adams, London, Sydenham Society, 1849, vol. 2, p. 774.

3. Celsus, A. C.: Of Medicine in Eight Books, translated by J. Greive, London, D. Wilson & T. Durham, 1756.

4. Albucasis: De chirurgia, Oxford, Clarendon Press, 1778, vol. 2, p. 23; cited by Mukherjee.[72]

5. Aeginata, P.: The Seven Books of Paulus Aeginata, translated and edited by F. Adams, London, Sydenham Society, 1846, vol. 2, p. 28.

Suśrutian teachings on cauterization and on liver therapy in night blindness have been reflected in the teachings of Hippocrates.

In speaking about the efficacy of cauterization, Suśruta said:[10] "With regard to surgical treatment, actual cautery is said to be superior to caustics, inasmuch as diseases treated with the actual cautery do not reappear, and because it can cure diseases which are incurable by medicines, instruments and caustics."

With this Suśrutian principle may be compared the following aphorism of Hippocrates, who taught:[2] "Those diseases which medicines do not cure, the knife cures; those which iron cannot cure, fire cures; and those which fire cannot cure, are to be reckoned as wholly incurable."

Again in connection with the treatment of night blindness, one finds that Suśruta[11] as well as Hippocrates[2] advocated liver therapy, which unmistakably shows the similarity of their teachings in this respect.

The observations of Vagbhatta[12] (about 700 A. D.), Chakrapani Datta[8] (1060 A. D.), Baizeau[13] (1861), Bitot[14] (1863), Blessig[13] (1866), Zak[15] (1917; 1920), Hift[15] (1918), Meisner[15] (1919), Mori[15] (1923), Pillat[16] (1929), Aykroyd[17] (1930), Spence[18] (1931), Jeans and Zentmire[19] (1934, 1936 and 1937), Park[20] (1935 and 1936),

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6. Rhazes: Tractatus nonus ad regem almansorem de curatione morborum particularium: opusculum huic szculo accommodatissimum, Paris, apud S. Colinœum, 1534.

7. Vagbhatta: Astangahridaysamhitā, edited by Anna Moreswar Kunte, Bombay, Nirnaya Sagara Press, 1912.

8. Chakrapani Datta: Netraroga Cikitsa, in Chakradatta, edited by Jibananda Vidyasogara, Calcutta, Kavyaprakasa Press, 1872.

9. Sārangadhara: Sārangadhara Samgraha, chap. 3, verse 8.

10. (*a*) Suśruta Sámhitā,[1] Sūtrasthāna, chaps. 11 and 12. (*b*) Hoernle, A. F. R.: Translation Bibliotheca Indica.

11. Suśruta Saṁhitā,[1] Uttara Tantra, chap. 17, verses 20-25.

12. Vagbhatta.[7] Suśruta Saṁhitā,[1] Sūtrasthāna, chap. 15; Uttarasthāna, chaps. 8-16.

13. Cited by Blegvad, O.: Xerophthalmia, Keratomalacia and Xerosis Conjunctivae, Am. J. Ophth. **7**:89, 1924.

14. Bitot: Memoire sur une lésion conjunctivale non encore décrite, Gaz. hebd. méd. et de chir. **10**:284, 1863.

15. Cited in Vitamins: A Survey of Present Knowledge, Medical Research Council, Special Report Series, no. 167, London, His Majesty's Stationery Office, 1932, p. 234.

16. Pillat, A.: The Frequency of Deficiency Diseases in the Eye Due to Lack of Vitamin A in a Military Camp in Peiping, Nat. M. J. China **15**:595, 1929.

17. Aykroyd, W. R.: Night Blindness Due to Vitamin Deficiency, Tr. Ophth. Soc. U. Kingdom **50**:230, 1930.

18. Spence, J. C.: A Clinical Study of Nutritional Xerophthalmia and Night-Blindness, Arch. Dis. Childhood **6**:17, 1931.

(Footnotes continued on next page)

Frandsen [21] (1935), Jeghers [22] (1937), Fridericia and Holm [23] (1925), Wolff [24] (1932) and others in connection with vitamin therapy in ocular diseases are in exact consonance with the teachings of Suśruta.[25] The views of Suśruta [26] regarding the efficacy of local application of vitamin A preparations for the treatment of various ocular diseases are upheld by the investigations of the modern ophthalmologists, namely, Balachovski [27] (1934), Kaptsiovskaya [28] (1934) and others.

The Suśrutian principles on cauterization [29] in ocular diseases have been ardently advocated and followed by Vagbhatta,[30] Sārangadhara [9] (1500 A. D.), Chakrapani Datta,[8] Coulter [31] (1936), Monbrun and Castéran [32] (1929), Fuchs,[33] Parsons,[34] May and Worth [35] and others.

Among the most enthusiastic exponents of Suśruta's principle of vascular obliteration in the treatment of various forms of keratitis [36] may

19. Jeans, P. C., and Zentmire, Z.: Clinical Method for Determining Moderate Degrees of Vitamin A Deficiency, J. A. M. A. **102**:892 (March 24) 1934. Jeans, P. C.; Blanchard, E., and Zentmire, Z.: Dark Adaptation and Vitamin A: New Photometric Technic, ibid. **108**:451 (Feb. 6) 1937.

20. Park, I. O.: Preliminary Observations on Vitamin A Deficiency as Shown by Studies with Visual Photometer, J. Oklahoma M. A. **28**:357, 1935; Further Studies on Vitamin A Deficiency as Shown by Studies with Visual Photometer and Clinically, ibid. **29**:129, 1936.

21. Frandsen, H.: Hemeralopia as Early Criterion of A-Avitaminosis and Clinical Symptoms and Treatment of This Disease, Acta ophth., 1935, supp. 4, p. 1.

22. Jeghers, H.: Night Blindness Due to Vitamin A Deficiency, New England J. Med. **216**:51, 1937.

23. Fridericia, L. S., and Holm, E.: Experimental Contribution to the Study of the Relation Between Night Blindness and Malnutrition: Influence of Deficiency of Fat-Soluble A Vitamin in Diet on Visual Purple in the Eyes of Rats, Am. J. Physiol. **73**:63, 1925.

24. Wolff, L. K.: On the Quantity of Vitamin A Present in the Human Liver, Lancet **2**:669, 1932.

25. Suśruta Saṁhitā,[1] (*a*) Uttara Tantra, chap. 17; (*b*) Sūtrasthāna, chap. 35.

26. Suśruta Saṁhitā,[1] Uttara Tantra, chaps. 9-19.

27. Balachovski, S.: Sur la possibilité d'insuffisances locales en vitamines, Presse méd. **42**:1404, 1934.

28. Kaptsiovskaya, R. S.: Clinical Characteristics of Carotene Used in Treatment of External Diseases of the Eye, Sovet. vestnik oftal. **5**:242, 1934.

29. Suśruta Saṁhitā,[1] Sūtrasthāna, chaps. 11 and 13.

30. Vagbhatta.[7] Suśruta Saṁhitā,[1] Sūtrasthāna, chap. 30.

31. Coulter, J. S.: Medical Diathermy, J. A. M. A. **106**:209 (Jan. 18) 1936.

32. Monbrun, A., and Castéran, M.: La haute fréquence en ophtalmologie, Paris, Masson & Cie, 1929.

33. Fuchs, E.: Diseases of the Eye, ed. 10, translated by E. V. L. Brown, Philadelphia, J. B. Lippincott Company, 1933, pp. 163 and 172.

34. Parsons, J. H.: Diseases of the Eye, ed. 7, London, J. & A. Churchill, Ltd., 1934, p. 213.

35. May, C. H., and Worth, C.: A Manual of the Diseases of the Eye, ed. 5, London, Baillière, Tindall & Cox, 1927, chap. 8, p. 126.

36. Suśruta Saṁhitā,[1] Uttara Tantra.

be mentioned such illustrious persons as Vagbhatta,[37] Janin [38] (1772), Chakrapani Datta,[8] Boury [39] (1743), Sārangadhara,[9] de Saint Yves [40] (1767), Scarpa [41] (1801), Bell [42] (1816), Gundersen [43] (1939) and others. Most of the findings of Bowman [44] (1849) in connection with his investigations on the anatomic structure of the cornea as well as the pathologic picture of corneal inflammation coincide exactly with those of Suśruta.[45] In other words, all these astute observers have discovered in the post-Suśrutian period what Suśruta discovered and taught in the prehistoric age.

Of course, improvements and modifications of many of the old methods based on Suśrutian teachings have been worked out and numerous new technics have been devised and introduced into the field of ophthalmic surgery. For instance, the principles of cataract surgery as originally taught by Suśruta [46] have undergone thorough and radical evolution and overhauling at the hands of Daviel,[47] Wright,[48] Smith,[49] Knapp,[50] Barraquer,[51] Verhoeff,[52] Elschnig,[53] Sinclair,[54] Török [55] and Pahwa [56] and others.

37. Vagbhatta.[7] Suśruta Saṁhitā,[1] Uttarasthāna, chap. 11.

38. Janin, cited by Gundersen.[43]

39. Boury, J. W., in Mauchart, J. D.: De maculis corneae earumque operatione chirurgica, apotripsi, Tubingen, typ. J. D. Bauhofi ac viduae Pflickii, 1743.

40. de Saint Yves, C.: Nouveau traité des maladies des yeux, Amsterdam, Arkstée & Merkus, 1767, pp. 171 and 178.

41. Scarpa, A.: Saggio di osservazioni e d'esperienze sulle principali malattie degli occhi, Pavia, Baldassare Comino, 1801, p. 106.

42. Bell, C.: Operative Surgery, ed. 2, Hartford, Hale & Hosmer, 1816, vol. 2, p. 90.

43. Gundersen, T.: Vascular Obliteration for Various Types of Keratitis, Arch. Ophth. **21**:76 (Jan.) 1939.

44. Bowman, W.: Lectures on the Parts Concerned in the Operations upon the Eye, and on the Structure of the Retina, London, Longman [and others], 1849.

45. (*a*) Suśruta Saṁhitā,[1] Uttara Tantra, chap. 12. (*b*) Mādhavkara: Pathology of Hindu Medicine, with Commentary of Vijayaraksita, ed. 5, p. 309.

46. (*a*) Suśruta Saṁhitā.[25] (*b*) Bidyādhar, N. K.: Suśruta and His Ophthalmic Operations, Arch. Ophth. **22**:550 (Oct.) 1939.

47. Daviel: Sur une nouvelle methode de guérir la cataracte par l'extraction du cristalin, Mém. Acad. roy. de chir. **2**:337, 1753. Translation by the late Dr. D. W. Greene, of Dayton, Ohio.

48. Wright, J. W.: Improved Operation for Extraction of Cataract, Columbus M. J. **3**:145, 1884.

49. Smith, H.: The Treatment of Cataract and Some Other Ocular Affections, Calcutta, Butterworth & Co., Ltd., 1928.

50. Knapp, A.: Report of a Second Hundred Successive Extractions of Cataract in Capsule After Preliminary Subluxation with Capsule Forceps, Arch. Ophth. **50**: 426, 1921; Late Results of Intracapsular Cataract Extraction, Tr. Ophth. Soc. U. Kingdom **45**:117, 1925.

51. Barraquer, J. A.: Phacoerisis, Arch. Ophth. **50**:307, 1921; **51**:448, 1922. Barraquer, J. A., and Anduyned: Un procédé d'extrême douceur pour l'extraction "in toto" de la cataracte, Clin. opht. **22**:328, 1917.

(Footnotes continued on next page)

Unfortunately, however, the history of ophthalmology in the prehistoric Suśrutian age has been vaguely and imperfectly understood, as would be inferred from the following few quotations:

Fuchs,[57] speaking about the treatment of night blindness, wrote: "So far as the treatment is concerned the eating of cooked liver as well as cod liver oil has stood in great and serviceable repute with people since time immemorial. In addition, one looks after improvement of the nutrition through a strengthening diet and energizing drugs."

Gifford,[58] in his excellent treatise, stated: "Liver and liver therapy have long been used in hemeralopia with good results, because of the richness of this tissue in vitamin A."

Again, Gifford [59] remarked: "Copper sulphate, which has been used for thousands of years, is still the drug which by its combined astringent and bactericidal effect, is apparently of most value in trachoma."

Gundersen (1939) in a learned article wrote: [60] "The observation that reducing the blood supply has a healing effect on corneal lesions was first made almost two centuries ago. The use of astringents or of surgical treatment to effect this result was advocated then more universally and with greater clarity than it is today."

In order that the history of the evolution of this most important branch of medical science be understood and recorded in its true perspective, the scientific worker must report the successive phases of development of the scientific principles in a truly chronologic order. In attempting to record, therefore, the teachings of Suśruta on ocular therapy, my object is not only to contribute an unwritten and important chapter in the history of the evolution of ophthalmic surgery but to show how far the Suśrutian teachings have influenced and dominated the evolution of the science of ophthalmology.

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52. Verhoeff, F. H.: Improved Capsule Forceps for Intracapsular Cataract Extractions, Tr. Am. Ophth. Soc. **14**:489, 1916.

53. Elschnig, A.: Die intrakapsuläre Starextraktion, Berlin, Julius Springer, 1932.

54. Sinclair, A. H. H.: A Preliminary Note on the Intracapsular Extraction of Cataract with Specially Designed Forceps, Brit. M. J. **2**:661, 1924; Intracapsular Extraction of Cataract, with Some New Instruments and Their Use, Tr. Ophth. Soc. U. Kingdom **45**:127, 1925; Presidential Address, ibid. **52**:lvii, 1932.

55. Török, E.: Extraction of Cataracts in the Capsule by a Slight Modification of von Graefe Method, Ann. Ophth. **25**:712, 1916.

56. Pahwa, M.: The Intracapsular Extraction in Senile Cataract by Tumbling of the Lens, Antiseptic **36**:544, 1939. In this paper Dr. Pahwa is reported to have done as many as 147,461 cataract extractions by the intracapsular operation.

57. Fuchs,[33] chap. 16, pp. 412-413.

58. Gifford, S. R.: A Handbook of Ocular Therapeutics, ed. 2, London, Henry Kimpton, 1937, chap. 3, p. 68.

59. Gifford,[58] chap. 7, pp. 173-174.

60. Gundersen,[43] p. 77.

GENERAL CONSIDERATIONS

Not unlike the modern ophthalmic surgeon, Suśruta advocated both local treatment of the eye and treatment of any constitutional disease predisposing to and aggravating the ocular condition. Elimination of bodily toxins by purgatives and emetics in order to pacify the deranged bodily humors was rightly stressed as a preliminary to all varieties of therapeutic measures. According to Suśruta's teaching,[61] the simple maxim to be followed in ocular therapy is to avoid and guard against the primary specific factors predisposing to the disease. Special remedial measures are to be used for the deranged bodily humors and to combat the concomitant complications. This maxim is indeed safe, scientific and in agreement with modern practice.

Suśruta rightly warned the physician to guard against predisposing etiologic factors; e. g., he clearly laid down the rule that the occurrence of ophthalmia (*abhiśyanda*) should be promptly noted and the disease treated at once, as otherwise it will lead to keratitis, uveitis and various other intraocular inflammations.

Like the modern ophthalmologist, Suśruta advised washing the organ with decoctions of antiseptic, astringent and antiphlogistic drugs. The use of compresses (*svedas*), medicinal poultices (*upahāhas*), soothing oleaginous medicaments (*snehanas*), ointments (*anjanas*), medicinal plasters (*pralepas*), fumigation and inhalation of medicinal fumes or vapors (*dhūma*), snuff (*nasya*) and medicated antiseptic gargles (*kavalas*) and the sprinkling of or filling of the eye with lotions (*aschyōtana*) are among the general measures prescribed by Suśruta in the treatment of ocular diseases. He also advised cauterization, physical therapy (massage—*pratisārana*) and venesection by means of instruments or the application of leeches, according to the indications of the disease. Among the special measures, Suśruta invariably prescribed the administration of *tarpana*[62] (soothing medication) and *putapāka*[62] (special form of chemotherapy). He was the first scientist to recognize the importance of vitamins and to introduce vitamin therapy, both local and general, in the treatment of ocular diseases.[63] The modern ophthalmologist will, however, be surprised to learn that it was Suśruta who first prescribed internal administration of cooked liver and the application of an ointment containing liver extract in the treatment of night blindness. Explained in the light of modern science, this means that vitamin A was given for the amelioration of night blindness. In addition, Suśruta well knew that most of the ocular diseases, such as xerosis conjunctivae, are due to avitaminosis; hence he advised the

61. Bidyādhar,[46b] p. 555.
62. Suśruta Saṁhitā,[1] Uttara Tantra, chap. 18.
63. Suśruta Saṁhitā,[1] Sūtrasthāna, chaps. 45 and 46.

consumption of sufficient quantities of milk, butter, *ghrta* (clarified butter) and fresh fruits to insure a proper amount of vitamins.

As is thoroughly manifest from his writings on the pathologic process and the treatment of inflammatory conditions in general, Suśruta definitely mentioned the use of antiseptic (*viśa-hara*) and antiphlogistic (*vranaśodhaka*) medicaments, which shows that even in preantiseptic days bacterial infection and sepsis were combated by means of the application of decoctions of antiseptic, astringent and antiphlogistic drugs and that ulcers and inflammatory conditions in general were treated as successfully and as efficiently as by the modern ophthalmic surgeon.

Suśruta was the first to conceive of the deleterious and baneful effects of vascularization of the corneal tissues. He therefore rightly taught that devascularization helps greatly to clear up corneal inflammations more rapidly. He effected vascular obliteration principally by means of his scarifying ointment (*lekhana anjana*), although he also employed astringent ointments and surgical measures.

The use of cautery in the field of ophthalmic surgery was first demonstrated by Suśruta, who well knew the remarkable efficacy of cauterization in the treatment of many of the obstinate ocular diseases. Since then the practice of cauterization in ophthalmic therapeutics has been universally in vogue. Suśruta practiced both thermocautery (*agni-karma*) and caustic cautery (*kśāra-karma*).

Suśruta's ingenious classification of the ointments (*anjanas*) into oleaginous (*snehana*), healing (*rōpana*), scarifying (*lekhana*) and invigorating (*prasādana*), depending on the respective medicinal ingredients characteristic of each *anjana,* exhibits the highest evolution of his surgical skill. He used to accomplish by means of his *lekhana anjana* what the modern surgeon does by the help of surgical appliances.

Among the measures used for physical therapy, massage (*pratisārana*) was often employed by Suśruta in the treatment of chronic inflammation of the meibomian glands, of corneal opacities, of pterygia (*armans*) and of diseases affecting the inner surface of the eyelids. The massage was generally done with a medicated compound; e. g., a compound of honey and sodium chloride was rubbed on the site of operation after excision of a *parvanikā* (a copper-colored phlyctenule at the limbus), or a pulverized compound of equal parts of potassium carbonate, *trikatu* (Piper longum, dry ginger and black pepper) and sodium chloride was rubbed on the site of the pterygium after its excision, while in the treatment of diseases of the inner eyelid, such as papillary conjunctivitis and trachoma, the parts were rubbed with a compound of equal parts of *manahsila* (realgar), *kasisa* (ferrous sulfate), *trikatu* (red pepper, ginger and black pepper), *anjana* (black antimony), *saindhava* (salt [sodium chloride]) and *swarna-makśikā* (copper pyrites) finely pounded together.

Among the substances used by Suśruta for controlling[64] intraocular hemorrhage, the most important was mother's milk. He well understood the hemostatic properties of the breast milk, which he used principally in ocular practice. Indeed, mother's milk was the epinephrine of the preepinephrine days, and it thus formed an effective weapon in the armamentarium of the ophthalmic surgeon of that period. Besides employing mother's milk for its hemostatic properties, Suśruta frequently utilized it in most of the ocular diseases as an eye wash along with medicinal decoctions as well as in the composition of various ocular medicaments. Suśruta also freely prescribed cow's milk, goat's milk, old *ghrta* (clarified butter), butter, honey, sugar cane juice and lemon juice for consumption by the patient to insure a proper amount of vitamins.

The use of the magnet was known to the Hindu surgeons of ancient India, and in the Suśrutian age it was used to extract foreign bodies, such as particles of iron, from the eye.[65]

Suśruta's ocular materia medica is exhaustive, consisting as it does of metals and metallic salts and of drugs culled from the vegetable kingdom as well as mineral salts and jewels. To Suśruta, indeed, ophthalmic science is indebted for the introduction into ocular therapeutics of the most important medicaments, which have remained as useful and as valuable today as they were in the Suśrutian period.[25b] Among the higher metals may be mentioned: gold, compounds of which were extolled by Suśruta as highly efficacious in ocular diseases owing to their mild antiseptic, astringent, tonic and invigorating properties; silver, the salts of which are astringent, demulcent and reparative in action, and copper, compounds of which, including copper sulfate, owing to their astringent, antiseptic and scarifying (*lekhana*) properties, have stood the test of time and are still used as valuable ocular medicaments in the treatment of trachoma and many other ocular diseases. The other metals and their salts used by Suśruta in the treatment of ocular diseases are: bell metal (bitter, antiseptic and scarifying); iron and its salts, i. e., ferrous sulfate (astringent, mild antiseptic and scarifying); zinc (*zasadam*—astringent); mercury (*pārada*[66]—antiseptic and invigorating to the eyesight), and stannum (*rangam*—antiseptic and tonic). In addition to these, *samkha* (conch shell), *samudraphena* (Sepia officinalis), marine oyster-shell, pearl, ruby, coral, diamond, *vaidurya* (lapis lazuli), antimony, *asmantaka* (jewel) and *sphatika* (crystal) were used for their antiseptic, sedative, scarifying and tonic actions.

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64. Suśruta Saṁhitā,[25a] verse 61. Bidyādhar,[46b] p. 553.

65. Vaiśesikā Darśanam, chap. 5, Āhnika 15. Suśruta Saṁhitā,[1] Sūtrasthāna, chap. 27; cited by Mukherjee.[72]

66. Sen Gupta Kaviraj: Materia Medica of the Hindus, ed. 9, 1934, pp. 324-339.

The other drugs of vegetable, animal and mineral origin were most scientifically classified by Suśruta into groups on the basis of their tastes (*rasa*) and pharmacologic actions.[67]

Under drugs of the bitter (*tikta*) group he included *haridrā* (Curcuma longa), *dāru-haridrā* (Berberis asiatica), *rasānjana* (extract of Berberis asiatica), *kantakāri* (Solanum xanthocarpum), *brhati* (Solanum indicum), *mūdgaparni* (Phaseolus trilobus) and *brāhimi* (Enhydra fluctuans), all of which are antiseptic, antiphlogistic and tonic and reparative in their pharmacologic actions.

Under drugs of the sweet (*madhūra*) group he included *yastimadhu* (Glycyrrhiza glabra); *poundariyaka* (root stock of Nymphaea lotus); *zivanti* (Celtis orientalis); *drākśa* (grape—Vitis vinifera); *piyālā* (Buchanania latifolia); breast milk, cow's milk and goat's milk; butter and *ghrta* (clarified butter); bone marrow; barley, wheat and sugar cane juice and honey. These are demulcent, reparative, antiseptic, sedative and tonic and therefore highly useful in ocular diseases.

Drugs of the saline (*labana*) group included sodium chloride and potassium bicarbonate (*yavakśāra*). These are antiseptic, cleansing and mildly antiphlogistic.

Under drugs of the astringent (*kaśāya*) group Suśruta included *lodhra* (Symblocos racemosa), *triphala* (Terminalia chebula, Terminalia belerica and Embilica officinalis), *tagarapādukā* (Valeriana hardwicitrii), *kataka* (Strychnos potatorum), *priyangū* (Aglaia roxburghiana), *śūniśannaka* (Blepharis edulis) and *pālankyā* (Spinacia oleracea). These drugs are astringent, antiseptic, regenerative, scarifying and antiphlogistic in their action.

Drugs of the pungent (*katu*) group included *devadāru* (Pinus deodara), *kūstha* (Saussurea lappa), *mūstaka* (Cyprus rotundus), *pilū* (Salvadora persica) and *nirgūndi* (Vitis trifolia). These are mildly antiseptic and rubefacient in action.

Suśruta not only insisted on drug therapy (both local and general) in the treatment of ocular diseases but laid particular stress on diet suitable to the particular type of the disease concerned. He therefore invariably prescribed liquid diet prepared from barley and milk and light solid articles prepared from wheat and butter or *ghrta* (clarified butter). He also laid emphasis on the consumption of sufficient quantities of milk along with sugar cane juice or honey. Meat, fish, liver of goat and eggs were also prescribed. Among the vitamin-containing fruits, oranges, lemons, pineapples, grapes and bananas were mostly given. Carrots (*gārar*), radishes (*mūlaka*), brinjal*(*bartāku*), *sobhānjana* (Moringa pterygosperma), *agastipūspa* (Agati grandiflora) and spinach (*pālankyā*), all edible vegetables, were also included in the dietary of the patient.[68]

67. Suśruta Saṁhitā,[1] Sūtrasthāna, chap. 42.

68. Suśruta Saṁhitā.[25a] Sen Gupta Kaviraj,[66] pp. 381-406.

TECHNIC OF SPECIAL THERAPEUTIC MEASURES

Before describing the Suśrutian methods of ocular therapy, I shall give a brief explanatory review of the technics of the different therapeutic measures employed by Suśruta in combating diseases of the eye, namely, the use of *tarpana* and *pūtapāka* and of *aschyōtana* (eye lotion) and *pariseka* (eye wash), as well as the technic involved in the preparation and application of the different varieties of *anjanas* (ointments).

Suśruta's instruction for *tarpana* follow:[62] After the system has been thoroughly cleansed, both internally and externally, by means of purgatives, emetics, errhines and venesection and after the food has digested, *tarpana* should be administered by the physician either in the forenoon or in the afternoon, but preferably in the morning, at an auspicious hour. The patient should lie flat on a clean bed in a chamber which is not exposed to gusts of wind or to the glowing rays of the sun. The chamber should be so cleansed that there is not a particle of dust in the atmosphere. The eye socket, having been cleansed aseptically, should be evenly covered with a paste made of powdered *māsá* pulse (Phaseolus radiatus) and sterile water, the plaster being so applied as to be smooth, uniformly thick and compact. After this, a sufficient quantity of the transparent upper layer of *ghrta* (clarified butter), either alone or combined with medicinal ingredients, mixed with a similar quantity of clean lukewarm water should be poured slowly on the eye socket up to the eyelashes and retained in the case of a healthy person as long as it would take to count 500 syllables; in the case of a person suffering from ocular disease due to lymphatic derangement, 600 syllables; in the case of a person suffering from a biliary type of ocular disease, 800 syllables, and for ocular diseases of the neural type, 1,000 syllables. After this, the *ghrta* should be carefully drained out of the eye socket, which again should be cleansed by the surgeon.

According to Suśruta, the use of *tarpana* is indicated for blurred vision, dryness of the eye, absolute absence of lacrimation, xerosis of the lid, depilation of the eyelashes, hardness of the eyelid and an acute disease of the eye. It should not be applied on a cloudy day or when the atmosphere is very hot or very cold; neither should it be used if the patient is apprehensive.

Suśruta, quoting from Jejjatācharya, stated that one day's application of *tarpana* is sufficient for a slightly aggravated condition of the eye, three days for a moderately diseased condition and five days for an acutely aggravated condition.

The characteristic features of satisfactory use of *tarpana* as described by Suśruta are: sound sleep, comfortable waking, absence of inflammatory exudation, clearness of vision, feeling of lightness of the organ and perceptible amelioration of the ocular disease. Symptoms

of excessive use of *tarpana* are: heaviness of the eye, blurred vision, excessive glossiness of the organ, lacrimation and an itching sensation, sliminess of the organ and aggravation of the disease. Symptoms arising from the improper use of this treatment are: dryness of the organ, excessive lacrimation, inability to see clearly and aggravation of the disease.

Suśruta gave the following instructions for the use of *putapāka*:[62] The diseased organ should be compressed with a piece of clean linen soaked in warm water, both at the beginning and at the end of the treatment. When the treatment is completed, fumigation of the organ with medicinal ingredients should be prescribed for a disease characterized by derangement of the lymphatic humor.

Two *palas* (about 24 drachms [93.6 Gm.]) of cleansed meat made into a paste and 1 *pala* of the medicinal ingredients (according to the nature of the *putapāka*) are mixed with 8 *palas* of liquid ingredients (milk or a decoction of astringent drugs, etc.) and made into a ball; this should be covered with the leaves of one of the following plants: plantain, *eranda* (Ricinus communis), *kūmuda* (Nymphaea stellatta) or *padma* (Nelumbium speciosum). The ball is then plastered or coated with a uniform and compact layer of clay, which should be scorched in the fire of catechu wood, *kataka* (clearing nut tree) wood, *vāsaka* (Adhatoda vasica) or *Asmantaka* (wood sorrel) wood or in the fire of dried cow dung cakes. When sufficiently and uniformly burnt, the ball should be taken out of the fire and broken and its contents collected into a piece of clean sterilized lint. The liquid chemical extract from the ingredients should be squeezed out from the lint, collected in a clean pot and poured on the affected eye in the manner described for *tarpana*.

For diseases of the eye caused by biliary derangement or diseases of the blood, Suśruta wisely prescribed therapy with cold *putapāka* or the application of cold *tarpana;* for those caused by nervous disorders of lymphatic derangement lukewarm *putapāka* or *tarpana* are to be employed.

Properly administered, putapāka should allay inflammation, chemosis, intraocular pain and itching sensation; it should diminish mucoid exudation and lacrimation, and it should also successfully effect vascular obliteration in vascularization of the cornea and sclera in which case *lekhana* (scarifying) *putapāka* is administered. Judiciously applied, *tarpana* and *putapāka* bring health and happiness to the diseased eyes.

According to Suśruta, *putapākas* as well as *tarpanas* are of three classes, according to the characteristic medicinal property they possess; namely, *snehana* (oleaginous), *lekhana* (scarifying) and *rōpana* (healing).

Indications for the different varieties of *putapāka* are given by Suśruta as follows: *Snehana* (oleaginous) *putapāka* is indicated when the affected eye is very dry (xerosis conjunctiva,); *lekhana* (scarifying) *putapāka,* when the affected organ is highly vascularized (episcleritis, corneal vascularization, conjunctival injection and vascularized pterygium) and therefore soft and filled with slimy exudation, and *rōpana* (healing) *putapāka,* when the conjunctiva or cornea is in an ulcerated or highly inflammatory condition.

The composition of the different varieties of *putapāka* is described by Suśruta as follows: *snehana putapāka* is composed of fleshy meat (flesh of animals frequenting marshy places); lard, marrow and fat; drugs of the sweet *(madhūra)* group, i. e., *yastimadhu* (glycyrrhiza,), *padmakāstha* (Prunus pudum), *gulancha* (Tinospora cordifolia), and *vansalōchana* (the manna of the bamboo plant). This compound should be retained as long as it takes to utter 500 syllables.

Lekhana putapāka is composed of flesh and liver of wild animals, such as deer, and drugs possessing scarifying properties, namely, powders of steel, copper, conch sheel, *vidrūma* (coral), *saindhava* salt (sodium chloride), *samudraphena* (Sepia officinalis), *kasisa* (ferrous sulfate), and *sauvirānjana* (antimony sulfide) mixed with the cream of curd. This *putapāka* should be retained only as long as it takes to utter 100 syllables.

Rōpana putapāka is composed of breast milk, flesh of wild animals, such as deer, honey, *ghrta* (clarified butter) and drugs of the bitter group, namely, *kantakāri* (Solanum xanthocarpum), *chiretta* (Swertia chirata), *gūlancha* (Tinospora cordifolia) and *nimva* (Melia azadirachta). These drugs are highly useful as demulcent, rubefacient and reparative and soothing medicament. This type of *putapāka* should be retained three times as long as *lekhana putapāka.*

One day is sufficient for the use of *lekhana putapāka* and two for *snehana putapāka,* while three days is rightly prescribed by Suśruta for the administration of *rōpana putapāka.*

During the period of therapy with *putapāka* the patient should observe a strict regimen of diet and conduct. After the application of *putapāka* he should be advised to restrain himself from looking at strong light, burning fire or such other luminous objects or from exposing the eye to strong wind. The complications arising from injudicious use of *putapāka* are to be treated by the application of a soothing ointment, by compresses and by the use of antiseptic and antiphlogistic eye washes.

Suśruta [62] ingeniously classified *aschyōtanas* and *parisekas* into three varieties: namely, *snehana, lekhana* and *rōpana* according to their characteristic properties. The medicinal ingredients possessing the characteristic properties of the different varieties are employed in their

preparations. Not unlike *putapāka,* the doses of different types of *aschyōtana* vary; for instance, 7 to 8 drops of *lekhana aschyōtana* are used, 10 drops of *snehana aschyōtana* and 12 drops of *rōpana aschyōtana.* The duration of treatment with *pariseka* (eye wash) is double that with *putapāka,* or it should be regularly employed until the disease is cured. In general, the different varieties of *aschyōtana* and *pariseka* are to be employed according to the discretion of the physician with particular reference to the nature of the ocular disease.

Suśruta prescribed *lekhana aschyōtana* for *kaphaja* diseases of the eye (those due to lymphatic derangement); *snehana aschyōtana* and *pariseka* for *vataja* diseases (nervous disorders), and *rōpana aschyōtana* and *pariseka* for *raktaja* diseases (blood disorder).

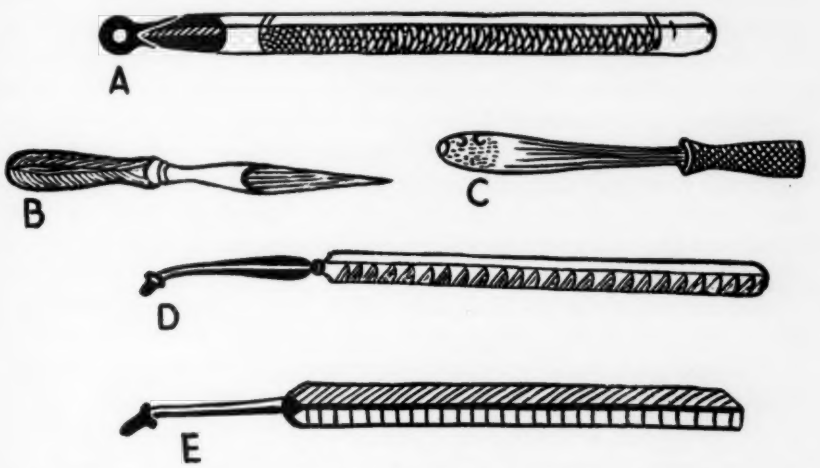
Suśruta's teachings with regard to the technic of the preparation and application of *anjanas* (ointments) and collyrium probes follow:[62] He advised application of *anjanas* after the system has been cleansed both externally and internally. Anjanas are classified by Suśruta into *snehana* (oleaginous), *rōpana* (healing, *lekhana* (scarifying) and *prasādana* (invigorating to the eye sight), according to their characteristic and distinguishing properties. They are further subclassified into *gutikā* (tablets), *rasakriyā* (liquid) and *chūrna* (powder), according to their potency and form, each of the preceding *anjana* being stronger than the one succeeding it. That is to say, *gutikā anjana,* being the greatest in efficiency, is used in the treatment of the most acute forms of the disease, *rasakriyā anjana* is used in moderately aggravated conditions, while *chūrna anjana,* being the least potent, is prescribed for mild forms of the disease.

The composition and characteristic properties of the different varieties of *anjana* as described by Suśruta follow: *Lekhana anjana* is prepared with drugs of one or more flavors, except the sweet (*madhūra*) ones, mixed with equal parts of powdered *samkha* (conch shell), *samudraphena* (Sepia officinalis), marine oyster shell, crystal, *asmantaka* (jewel), powdered iron, copper and antimony. The *lekhanna anjana,* by virtue of the characteristic properties of its constituents, exercises a scarifying and destructive as well as a fibrolytic action on vascularization of the ocular structures. It is therefore best used, according to Suśruta, for the various forms of keratitis accompanied with vascularization, for episcleritis and for various forms of fibrovascular *armans* (pterygia).

Rōpana anjana is made from drugs with a bitter and astringent taste, mixed with a moderate quantity of clarified butter. It is therefore demulcent, rubefacient and most efficacious for healing purposes, as in the treatment of corneal ulcers and purulent ophthalmia. Owing to the presence of *sneha* (i. e., clarified butter), it is cooling and sedative in its effect and reparative in its action.

Prasādana, or *snehana, anjana* is prepared from drugs with a sweet *(madhūra)* taste mixed with a sufficient quantity of clarified butter. Special sight-invigorating substances, such as gold, jewels and milk, are added. This *anjana,* being demulcent and having a vitamin content owing to the *ghrta* (clarified butter, etc.), imparts tone to the eye and increases visual acuity.

Suśruta described the materials of the vessel for the preservation of the *anjana* as well as that of the collyrium probes as follows: Gold vessels are required for storing *anjanas* compounded with sweet drugs; silver ones for *anjanas* made with acid drugs; goat's horn vessels for saline ointments; copper or iron vessels for astringent ointments; *vaidurya* (lapis lazuli) vessels for sour or pungent ointment, and bell metal vessels for bitter *anjanas.*



A, Suśruta's collyrium salākā (rod) made of gold, silver, steel, bell metal, copper or horn. The instrument is 8 *angulis* (fingers) long (from Mukherjee,[72] p. 161). *B,* Suśruta's *vetasapatra jantra.* It is a sharp cutting instrument made of steel, the blade and handle being of equal length, measuring 4 *angulis* (from Mukherjee,[72] p. 264). *C,* Suśruta's *jamvavaustha jantra.* This instrument is made of steel, one end being shaped like the fruit of Eugenia jambolanum, whence its name. It may be made of any length required by the surgeon and is utilized for applying actual cautery (from Mukherjee,[72] p. 160). *D,* Suśruta's gold cautery, made of any suitable length desired by the surgeon, the end tapering and shaped like the *ankuśa* (elephant driver's goad) (from Mukherjee,[72] p. 159). *E,* Chakrapani Datta's gold probe made in various lengths according to the requirements of surgeons, the end tapering like a salākā (probe) (from Suśruta Samhita,[1] Sustrasthāna, chap. 7).

As for the collyrium probes [69] (*A* in the accompanying illustration), Suśruta recommended probes of different metals for different types of *anjanas;* namely, a probe *(salākā)* of steel, bell metal or copper for

69. Suśruta Saṁhitā.[62] Mukherjee,[72] pp. 160-161.

lekhana collyrium and one of gold, silver or horn for *rōpana* (healing), *snehana* (oleaginous) and *prasādana* (invigorating) ointments. The probe should be 8 *angulis* (fingers) long, the end of the rod terminating in a bud-shaped (pealike) ball of the size of a pea. It should be well polished, slender at the middle and so fashioned as to be easily manipulated. A collyrium *salākā* made of copper, precious jewels (such as lapiz lazuli) and horns is recommended by Suśruta as being highly beneficial.

In connection with the collyrium probes, the views of the Hindu, Greek and Roman surgeons of the post-Suśrutian periods are of scientific interest.

According to Chakrapani Datta,[70] for the application of *lekhana anjana* the probe should be made of copper and 10 angulis (fingers) in length. It should be made comparatively thinner at its middle to facilitate a firm grasp by the surgeon, and the ends should be bud shaped. For the application of *rōpana* collyrium, a probe of steel is recommended, while for *prasādana* (invigorating) collyrium for the improvement of visual acuity, a probe of gold or silver should be used.

Sārangadhara said[71] "The collyrium probe should be made of stone or metal, 8 *angulis* (fingers) long, the ends being smooth and round like a common pea. For *lekhana* collyrium, a copper, iron or stone probe is to be recommended, whereas for application of *rōpana* collyrium the surgeon should use the tip of the finger with efficacy."

Mukherjee wrote:[72] "Specillum with two olivary ends formed a variety of probe of the Greek and Roman surgeons . . ."

A single probe for the application of semisolid medicaments is contained in the outfit of the oculist of Rheims, in the museum at St. Germain-en-Laye, and is depicted by Milne.[73]

The mode of application of *anjanas* is described by Suśruta as follows: With his left hand the surgeon should evert the eyelids in a slanting fashion, and holding the collyrium *salākā* (probe) with his right hand he should apply the *anjana* carefully and lightly, constantly moving the rod from the mesial angle of the eye *(kaninikā)* to the exterior corner *(apānga)*, this process being repeated two or three times, according to the indications of the condition. The surgeon should apply the *anjana* with his clean finger when it is necessary to put it on the outside of the eyelid. In no case should he thickly anoint the collyrium in the corners of the eye, lest the corners be injured.

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70. Chakrapani Datta,[8] Aschyōtana Chikitsā (Treatment of Eye Diseases).

71. Sārangadhara,[9] chap. 3, verse 13. Yogaratnakara: Eye Diseases, p. 823.

72. Mukherjee, G. N.: Surgical Instruments of the Hindus, Calcutta, Calcutta University, 1913, vol. 1, p. 163.

73. Milne, J. S.: Surgical Instruments in Greek and Roman Times, Aberdeen, 1907, pl. 11, fig. 5.

Suśruta forbade the applications of *anjanas* in the following instances: for persons suffering from fever and diseases of the head; for persons overcome with fear, sorrow or anger; for persons weeping or intoxicated; for persons suffering from retention of urine and stools, because the application of *anjana* under such circumstances would aggravate the deranged intraocular humors and bring about aggravation of inflammation, chemosis, lacrimation, pain, ciliary and conjunctival injection and dimness of visual acuity, and for persons suffering from insanity, as such a practice would tend to impair vision. He considered *anjanas* contraindicated for eyes subjected to dust or smoke, as they would cause irritation, ciliary and conjunctival injection, chemosis and lacrimation, all these in their turn leading to intraocular infection, such as iritis and iridocyclitis. He also considered them contraindicated immediately after the use of *nasya* (errhine), as they might increase inflammation, congestion and chemosis with attendant aching pain, and before sunrise, immediately after a head bath or on a very cold or very windy day. Similarly he stated that they were ineffective if the patient was suffering from indigestion, as the lymphatic system becomes engorged and therefore the excretory system of the eye becomes blocked.

Hence Suśruta advised the ophthalmic surgeon to exercise his utmost discretion, with due consideration of the vitality of the patient, the nature of the disease and the time for the application of the *anjanas,* and especially to observe all the aforementioned contraindications when applying a *lekhana anjana.*

The complications arising from the injudicious application of *anjanas* should be treated by application of antiseptic and antiphlogistic eye washes, by fumigations, and by the use of medicated gargles and snuffs, with due regard to the specific nature of the disease.

Suśruta described the symptoms of satisfactory, excessive and deficient use of *lekhana anjanas* as follows: Satisfactory application is evidenced by lightness of the organ, clearness of the cornea and sclera due to devascularization, improved visual capacity and absence of secretions and other complications. Excessive use is shown by looseness of the conjunctiva and sclera and marked scarification of the external coat of the eye, while deficient use is shown by the aggravation of the existing intraocular complications.

Suśruta rightly emphasized that deficient or improper use of *snehana* and *rōpana anjanas* is sure to be ineffective and abortive in producing the desired results. Hence he advised the ophthalmic surgeon to exercise his utmost discretion in the proper application of an *anjana.*

In addition to the foregoing therapeutic measures, Suśruta also included cauterization, fumigation, the use of snuff (*nasya*), venesection (*vyadhana*) by means of instruments as well as by application of leeches

and the use of a medicated gargle (*kavala*) in the therapeutic paraphernalia of the ophthalmic surgeon.

Among other measures for insuring the preservation of the general strength and resistance of the patient, Suśruta laid great stress on the diet and the conduct of the patient suffering from an ocular disease; for example, the nutrition of the system should be properly looked after by prescribing a strengthening, vitamin-containing diet and energizing medicaments. The hygiene of the affected eye should be maintained by warning the patient not to look at dazzling lights or the glaring sun and not to expose the eye to wind, dust or smoke. The attention of the patient must be given to maintaining a perfectly hygienic mental condition by remaining calm and quiet. Suśruta taught that constitutional diseases predisposing to the aggravation of ocular diseases should be promptly and effectively treated. He laid emphasis on cleansing the system both externally and internally by purgatives, emetics, oleaginous enemas, errhines and venesection, as well as by oral cleanliness. Any source of irritation to the eye, i. e., from the nose and teeth, should be speedily attended to.

Milk; butter, simple or medicated clarified butter; *triphala* (Terminalia chebula, Terminalia belerica and Embilica officinalis); *śatāvari* (Asparagus racemosus); *patōla* (Trichosanthes disica); *mūdga* (Phaseolus mungo); *amalaki* (Phyllanthus amblica), *java* (barley), curries prepared from edible vegetables, such as *zivanti* (Celtis orientalis), *jayanti* (Premna seratifolia), *sunisannaka* (Blepharis edulis), *tandūliya* (Amarantus spinosus), *bāstūka* (Chenopodium album), *mūlaka* (Raphanus sativus) and *agastipūspa* (Agati grandiflora), meat of birds, such as *tittira* (Francoline partridge), fowl and meat of wild animals, such as deer, are prescribed by Suśruta as invigorating foods for persons with ocular diseases.[74] Fish and liver of goat are prescribed for persons suffering from night blindness.

Concerning the technic of *vyadhana* (venesection), Suśruta advised that bloodletting be done either by puncturing with an instrument like the *vetasapatra* (*B* of the illustration) or by applying leeches. The various ocular diseases for which venesection was recommended by Suśruta have been enumerated by me[75] in a previous article as follows: *sirōt-pāta* (pannus), *sirā-harśa* (high grade pannus), *saśopha-akśipāka* (sympathetic ophthalmia with swelling), *śopha-akśipāka* (plastic endophthalmitis), *anyatō-vāta* (disease of neural origin), *pūyālasa* (acute dacryocystitis), *vāta-paryāya* (disease of neural origin), four types of *abhiśyanda* (ophthalmia) and four types of *adhimantha* (uveitis).

Suśruta's description of leeches[76] is so elaborate that the modern ophthalmic surgeon could study it with advantage and admiration.

74. Suśruta Saṁhitā[25a]; Sūtrasthāna, chap. 45.
75. Bidyādhar,[46b] p. 561.
76. Suśruta Saṁhitā,[1] Sūtrasthāna, chap. 13. Mukherjee,[72] pp. 280-288.

Suśruta clearly stated that of all forms for the extraction of blood, that by means of leeches is the mildest, and therefore that venesection by leeches is safely recommended for delicate, old and timid persons and for children and women. Suśruta described twelve kinds of leeches, of which six are poisonous and six nonpoisonous.

The poisonous leeches are: *krsnā,* which is the color of black ointment and characterized by a broad head; *karvūrā,* which is similar to an eel in length and has elevated stripes across the abdomen; *alagarddā,* which has large sides and a black mouth and looks as though it were covered with fine hairs; *indrāyūdha,* which is a rainbow-colored leech having rainbow-tinted longitudinal stripes on its back; *sāmūdrika,* which is dark yellow and has variegated flower-like spots on its body, and *gocandanā,* which has a small mouth and a bifurcated tail like the bull's scrotum. The nonpoisonous leeches are: *kapilā,* which has two sides of the color of orpiment and a smooth back the color of a green pea; *pingalā,* which is reddish brown, has a round body and moves more quickly than other leeches; *śankū-mūkhi,* which is liver-colored and sucks more quickly with its long sharp mouth than the other leeches; *mūsikā* (ratlike), which has the shape and color of a rat's tail and emits an offensive odor; *pūndarika-mukhi* (lotus faced), which has a mouth which looks like a lotus and is the color of a green pea, and *savarikā,* which is slimy, colored green like a lotus leaf, 8 *angulis* (fingers) long and is used in veterinary practice. The habitat of nonpoisonous leeches is in meadows and fresh water.

For the application of leeches, Suśruta directed that the patient lie down and that the part to be bled be rubbed dry with pulverized cow dung and sterile earth. The leech, after having been smeared with a paste containing turmeric, mustard and water, in order to excite it, should be thoroughly washed with water and then applied, its body having been covered with a piece of wet lint. When the leech has extracted the desired quantity of blood, a little salt should be sprinkled on its head to make it drop off at once. The leech bites should be smeared with honey along with antiseptic and astringent medicaments and duly poulticed.

As for cauterization in ocular therapy,[77] Suśruta distinctly mentioned the therapeutic efficacy of cauterization in the following diseases of the eye: the different types of uveitis (*adhimantha*), corneal ulcers, diseases of the eyelids, papillary conjunctivitis (*lagana*), chalazion, growths on the eyelids and trichiasis (*pakśma-kopa*). Like the modern ophthalmic surgeons, he clearly distinguished between medical cauterization and surgical cauterization in which a higher degree of heat is applied so as to cause destruction of tissue. He made use of both caustic cautery (*kśāra-karma*) and thermocautery (*agni-karma*). With regard to the therapeutic use of *kśāra-karma,* Suśruta wrote: "Of all cutting instru-

77. Suśruta Saṁhitā [10a]; Uttara Tantra, chaps. 9-16.

ments and their substitutes, caustics (or vegetable alkalis) are the most important, because by means of them deep and superficial incisions and scarifications may be made, derangements of the three humors may be rectified and some diseases can be treated with special advantage." The views of Suśruta regarding the therapeutic value of *agni-karma* are identical with those of Hippocrates, as well as with those of the Roman, Greek, Arabic and Hindu surgeons of the post-Suśrutian period.

For the application of the actual cautery, the following articles were used by Suśruta: *pippali* (Piper longum); goats' dung; teeth of a cow; *śara* (Saccharum munja); cautery probes of iron, gold, silver and copper, e. g., Suśruta's gold cautery and Chakrapani Datta's gold probe (*D* and *E* of the illustration); Suśruta's *jamvavaustha jantra* (*C* of the illustration); honey; treacle; *ghrta* (clarified butter), and oil.

According to Mukherjee:[78] "Both Aetius and Oribasius[79] represent goats' dung, pounded with vinegar, as being equally efficacious as the sinapism. Hippocrates[80] says that cauterization may be performed with box-wood spindles dipped in boiling oil. In Kordofan, are used 'El Kamaia,' primitive instruments for cauterisation, consisting of a piece of camels' or sheep's dung dried and impaled on a long thorn."[81]

Chakrapani Datta, a Hindu surgeon of ancient India, used a probe of gold (*E* of the illustration) for applying thermocautery to the hair follicles after the removal of the eyelashes in order to prevent the recurrence of trichiasis.[8]

The Arabian surgeons similarly used gold probes in the application of thermocautery. Albucasis,[4] like Suśruta, effected cauterization of the hair follicles in trichiasis by means of a probe of gold. Haly Abas and Rhazes also described the same procedure.[82]

The Greek surgeon, Aeginata, used a heated olivary probe or an aural probe for cauterization in trichiasis.[83]

In addition to the aforementioned therapeutic measures, Suśruta made use of medicated *dhūma* (fumigation), medicated *nasya* (snuff) and kavala (gargle).[84]

OUTLINE OF SUŚRUTA'S OCULAR THERAPEUTICS

Having discussed briefly the principles of Suśrutian ocular therapy and given a preliminary introduction to the subject, I shall now describe

78. Mukherjee,[72] p. 217.

79. Aeginata,[5] vol. 3, book 7, sect. 19.

80. Hippocrates,[2] vol. 2, p. 482.

81. Third Report of Medical Practices in Kordofan, Khartoum, Wellcome Research Laboratory.

82. Cited by Mukherjee,[72] p. 219.

83. Aeginata,[5] vols. 8-13.

84. Suśruta Saṁhitā,[1] Chikitsāsthana, chap. 40. Mukherjee,[72] pp. 141-145.

the medicinal treatment of the various ocular diseases as practiced and taught by Suśruta.

Much as I should like to deal with the subject elaborately, consideration of space prevents me from doing so. I shall therefore consider only the salient features.

In the following pages will be described the essential principles of Suśrutian ocular therapeutics for various types of *abhiśyanda* (ophthalmia) and *adhimantha* (uveitis), for *anyatō-vāta, vāta-paryāya* and other diseases of neural origin and for *amladhyuśita* (iritis), *suktika* (pinguecula), *dhūma-darśin* (amblyopia), *balāsa* (a speck the color of bell metal covered by vessels on the sclera), *pistaka* (a grayish-looking fleshy spot on the sclera), *praklinna-vartma* (acute gangrenous blepharitis), *pariklinna-vartma* (xerosis conjunctivae), *sirōt-pāta* (pannus) and *sirā-harśa* (high grade pannus), *arjuna* (blood red speck on the sclera), *śukra* (keratitis) and *ajakā-jata* (a sarcomatous growth invading the cornea). The treatment of night blindness, day blindness and retinitis will also be described.

The main points of Suśruta's treatment for *vātaja abhiśyanda*[85] (ophthalmia of neural origin) and *adhimantha* (uveitis) are as follows: The patient's body should be internally cleansed by full purging with the help of a *snehavasti* (oleaginous enema). The patient should also be treated with old and matured clarified butter. To the affected eye, which has been duly compressed, the following remedies should be administered according to the indications and gravity of the disease: *tarpana* and *putapāka,* fumigation, eye washes (*parisekas*), eye lotions (*aschyōtanas*), snuff (*nasya*) and medicated plaster (*pralepa*). Barley and milk, medicated *ghrta* (clarified butter) and other vitamin-containing medicaments should be taken internally. Venesection and cauterization should be resorted to according to the indications. Cauterization should be performed with either the *jamvavaustha jantra* (*C* of the illustration) or Suśruta's gold cautery (*D*), while venesection (*vyadhana*) should be done with a *vetasapatra jantra* (*B*).

Eye washes should be given with the decoction of drugs possessing sedative, antiphlogistic and antiseptic properties. The roots, leaves or bark of *eranda* plant (Ricinus communis) or the roots of *kantakāri* (Solanum xanthocarpum) should be boiled with the milk of a she-goat and made into a decoction. This should be utilized while warm for washing the affected organ.

A liquid compound prepared by boiling half milk and half water with equal parts of *saindhava* (sodium chloride), *vāla* (Pavonia odorata), *yastimadhu* (root of Glycyrrhiza glabra) and *pippali* (Piper longum) as well as *aschyōtanas* (eye drops) should be used for washing the eye.

85. Suśruta Saṁhitā,[1] Uttara Tantra, chap. 9.

A lint soaked in a lukewarm compound of equal parts of clarified butter, curd, fat and marrow should be applied over the affected organ, and a compound prepared from equal parts of milk, meat extract and porridge should be employed as a poultice. A liquid compound made by boiling the milk of a she-goat diluted with pure water with the addition of equal parts of *hrivera* (Pavonia odorata), *tagara* (Valeriana hardwicitrii), *manjistha* (Rubia cordifolia) and *ūdūmvara* (Ficus glomerata) may be highly beneficial for relieving pain in the eye. This should also be used as an *aschyōtana* (eye lotion).

Oleaginous medicaments chemically prepared by the admixture of drugs with sedative and antiphlogistic properties may be profitably used in the manner of *tarpana*. The use of *snehana putapāka* (oleaginous), *snehana* (oleaginous) *dhūma* (fumigation) and *oleaginous nasya* (snuff) is also prescribed. Oil properly cooked with *sthirā* (Desmodium gangaticum) and drugs of the *madhūra* (sweet) group, e. g., *yastimadhu* (glycyrrhiza) may be utilized as snuff.

An *anjana* prepared by mixing equal parts of *yastimadhu, haridrā* (Curcuma longa), *haritaki* (Terminalia chebula) and *devadāru* (Pinus deodara) with the milk of a she-goat is highly extolled by Suśruta for its efficacy in ophthalmia (*abhiśyanda*) as well as in uveitis (*adhimantha*). The other *anjana* which Suśruta used is *gutikā anjana,* which is prepared from *gairika* (red ocher), *saindhava* (sodium chloride), *pippali* (Piper longum) and *sūnthi* (dry ginger), the quantity of each succeeding drug being double that of the one preceding it. These ingredients are made into a paste with water and fashioned into a tablet (*gutikā*). This is beneficial in ophthalmia as well as in uveitis of neural origin. The use of *snehana* (oleaginous or demulcent) *anjana* is also recommended.

The patient should be given simple, light and nonstimulating diet. Vitamin therapy in the form of milk, medicated and plain, and medicated *ghrta* (clarified butter) is prescribed by Suśruta. After being fed, the patient should be given either a potion of clarified butter cooked with the decoction of *triphala* (Terminalia chebula, Terminalia belerica and Embilica officinalis) or simply old matured *ghrta.*

Suśruta advised the physician to adopt the aforementioned general remedial measures in the treatment of *anyatō-vāta* and *vāta-paryāya*—the two ocular diseases of neural origin. Before being fed, the patient with either of these diseases should be given medicated clarified butter and at the time of feeding he should have enough vitamins; i. e., he should take milk in sufficient quantity.

The different ways of preparing medicated clarified butter are described by Suśruta as follows: *Ghrta* may be boiled with two parts of the astringent juice expressed from *vriksādani* (Viscum album), *kapittha* (wood apple), *vilwa* (Aegle marmelos), *agnimantha* (Premna serati-

folia), *syonāka* (Bignonia indica), *pātoli* (Stereospermum suaveolens) and *gāmvāri* (Gmelina arboria), one part of the expressed juice of *karkata* (Rhus succedanea) and one part of milk; it may be cooked with a decoction of bark of *arjuna* (Terminalia arjuna), *ajmōda* (Seseli indicum), *sālincha* (Alternanthera sessilis) and milk, or it may be cooked with the decoction of *meśasrngi* (screw tree) and milk. This preparation should be taken before meals.

For the medicinal treatment of *súshkākshipāka* (plastic endophthalmitis), Suśruta [85] considered an *anjana* chemically prepared from a compound of equal parts of *saindhava* (sodium chloride), *devadāru* (Pinus deodara), *sūnthi* (dry ginger), juice of *mātūlūnga* (Citrus limmeta), clarified butter, breast milk and pure water to be very efficacious. He also regarded an ointment prepared by rubbing *sūnthi* with milk and *ghrta* or by mixing *sūnthi* and *saindhava* powder with the fat of an aquatic or nonaquatic animal and rubbing it on a stone slate to be highly efficacious. Eye washes composed of cold milk mixed with sodium chloride (*saindhava*) are beneficial in such cases. As alternative medicaments, eye washes prepared by boiling equal parts of *haridrā* (Curcuma longa) and *devadāru* (Pinus deodara) with milk and mixing the same with a small quantity of sodium chloride are also to be prescribed. The patient should also be advised to take clarified butter cooked with the drugs of the *jibaniya* (vitamin) group. The application of *tarpana* with *ghrta* cooked with vitamin-containing drugs, as well as snuff with the same, are also recommended as efficacious remedies in such cases.

Medicinal therapy for *pittaja abhiśyanda* (due to biliary derangement) and *pittaja adhimantha* is recorded as follows: [86] Venesection, purgatives, eye washes, medicinal plasters, snuff and *anjanas* and a nutritious and simple diet containing vitamins are among the general measures. Venesection is effected by puncturing a vein either in the temporal region or in the vicinity of the nose or by the application of leeches.

Clarified butter or goat's milk cooked with equal parts of *gūnza* (Abrus percatorius), *sāli* (Oriza sativa), *saivāla* (Pistia stratiotes), *śailaveda* (Coleus amboinicus), *dāru-haridra* (Berberis asiatica), *elā* (large cardamom seeds), *utpala* (Saussurea lappa), *lodhra* (Symblocos racemosa), *mūsta* (Cyprus rotundus), *padmapatra* (root of Aplotaxis auriculata), *śarkarā* (refined sugar), *darvamūta* (root of Andropogon nardus), *iksū* (sugar cane or its juice), *tāla* (Borassus flabellifer), *vetasa* (Calamus rotang), *padma-kastha* (Prunus pudum), *drākśa* (grape), honey, *yastimadhu* (glycyrrhiza), *raktachandana* (Pterocarpus santalius), breast milk, *haridra* (Curcuma longa) and *anantamūla* (Indian

86. Suśruta Saṁhitā,[1] Uttara Tantra, chap. 10.

sarsaparilla), the solid ingredients having been pulverized together, should be employed as a *tarpana,* as an eye wash or as snuff. As an alternative, all the preceding drugs, or as many of them as are available, should be boiled either with *ghrta* (clarified butter) or with goats' milk and used as a snuff.

All sorts of sedative measures tending to pacify biliary derangement should be adopted in the treatment of these diseases.

Suśruta prescribed *anjanas* made from various combinations of ingredients: from the expressed juice of *palāsa* (Butea frondosa) or *sāllaki* (Boswellia serrata) mixed with honey and sugar; from the decoction of *yastimadhu* (glycyrrhiza) or *pālindi* (Ipomoea turpethum) mixed with honey and sugar; from the decoction of *musta* (Cyprus rotundus), *samudraphena* (Sepis officinalis), *utpala* (Saussurea lappa), *vidanga* (seeds of Embelia ribes), *elā* (large cardamom seeds), *amalaki* (Embilica officinalis) and *pitasālaka* (Pentapera tomentosa) mixed with milk and honey; from equal parts of *tālisapatra* (Pinus webbiana), *elā* (large cardamom seeds), red ocher, conch shell and nalada (root of fragrant grass) mixed with breast milk; from powders or decoction of *dhātaki* (Woodfordia floribunda) and *syandana* (Dalbergia oujeinensis) mixed with breast milk; from pulverized gold mixed with breast milk; from honey mixed with the flowers of Butea frondosa; from lodhra (Symblocos racemosa), grape, sugar, *utpala* (Saussurea lappa), root of Glycyrrhiza glabra and *vacha* (sweet flag) mixed with breast milk; from the bark of *rōchana* (Mallotus philippinensis) mixed with breast milk, and from *vālā* (Pavonia odorata), red sandalwood, bark of Ficus glomerata and *samudraphena* (Sepia officinalis) mixed with breast milk and honey.

Eye drops to be used for this disease may be prepared by compounding glycyrrhiza, *lōdhra* (Symblocos racemosa), grape, sugar and *utpala* (Saussurea lappa) with breast milk, or by compounding *vālā* (Pavonia odorata), *gāmbhāri* (Gmelina arboria), *amalaki* (Embelica officinalis), *haritaki* (Terminalia chebula) or *katphala* (Myrica sapida) with pure water.

The medicinal treatment of iritis and *śūktikā* (pinguecula) as described by Suśruta is briefly outlined as follows: The general therapeutic measures should consist of the use of hot fomentations, and antiseptic and antiphlogistic eye washes, the application of *anjanas* and venesection by means of leeches. Among the special measures, Suśruta enjoined that the patient should take medicated *ghrta* (clarified butter), milk and other vitamin-containing foods.

In a case of *sūktikā* an ointment prepared with sedative drugs should be speedily employed. An *anjana* compounded from equal parts of *vaidurya* lapis lazuli), *sphatika* (crystal), mūktā (pearl), *śamkha* (conch shell), silver and gold and refined sugar and mixed with honey

is said to be specific for this disease. For a patient suffering from *dhūma-darśin* (amblyopia; smoky vision), in addition to the usual aforementioned therapeutic measures, vitamin therapy, such as medicated and simple clarified butter, milk, vitamin-containing fruits and the liver of goat, should be prescribed.

The medicinal treatment of *kaphaja abiśyanda* and *kaphaja adhimantha* (due to lymphatic derangement), according to the Suśrutian principles, is as follows:[87] During the acute stage the patient should be treated with venesection and the use of compresses, snuff, fumigation, eye washes, medicated plasters, medicated gargles, eye lotions or drops and bland *putapāka*. Cauterization should be resorted to according to the indications. After the patient has been purged for three days and the system cleansed both internally and externally, *ghrta* cooked with one of the bitter drugs should be given in the morning; the diet should consist of such food as will not upset the deranged intraocular and bodily humors.

Compresses prepared with a warm decoction made from equal parts of the tender twigs or leaves of *tagara* (Valeriana hardwicitrii), *kānchana* (Bauhinia variegata), *tūlasi* (Ocimum album), *vilwa* (aegle marmelos), *sālincha* (Alternanthera sessilis), *pilū* (Salvadora persica), *akanda* (Calotropis gigantea) and *kapittha* (Feronia elephantum) should be regularly applied. After this treatment a medicated plaster composed of *vālā* (Pavonia odorata), *sūnthi* (dry ginger), *devadāru* (Pinus deodara) and *kūstha* (Saussurea lappa) should be applied around the outer region of the affected eye.

As very efficacious ocular medicaments, Suśruta prescribed the following *varti anjanas* (ointment sticks): *anjanas* made from equal parts of *saindhava* (sodium chloride), *hingū* (asafetida), *triphala, yastimadhu* (glycyrrhiza), *pundariyakāstha* (root stock of Nymphaea lotus), *rasānjana* (extract of Berberis asiatica), *tuttha* (cupric sulfate) and powdered copper, all these having been mixed to a paste with water; from equal parts of *haritaki* (Terminalia chebula), *haridrā* (Curcuma longa), glycyrrhiza and *rasānjana* mixed to a paste water; from equal parts of *trikatu* (red pepper, ginger and black pepper), *triphala* (Terminalia chebula, Terminalia belerica and Embelica officialis) and *manahsilā* (realgar) mixed with water; from *vālā* (Pavonia odorata), *kustha* (Saussurea lappa), *devadārū* (Pinus deodara), pulverized conch shell, *ākanda* (Calotropis gigantea), *chitraka* (Plumbago zeylanica), *trikatū* and realgar mixed into a uniform and thick paste with water; from the flowers and fruits of *śajinā* (Moringa pterygosperma), *vrhati* (Solanum indicum) and *kantakāri* (Solanum xanthocarpum) with equal parts of *rasānjana,* sandalwood, sodium chloride, realgar, *haritāla* (orpiment) and

87. Suśruta Saṁhitā,[1] Uttara Tantra, chap. 11.

laśuna (Allium sativum) mixed with water, or from the flowers of *jāti* (Jasminum grandiflorum), *karanja* (Pongamia glabra) and *śajinā* and the fruits of *karanja* mixed with water. The *varti anjanas* are made by preparing thick pastes from the medicinal ingredients and shaping the pastes into the forms of a rod or stick (*varti*).

For the treatment of *balāsa* (a speck on the sclera the color of bell metal, covered by vessels) Suśruta [87] stated that the patient's system should be cleansed both internally and externally by emetics, purgatives, enemas, oleaginous medicament and bloodletting, followed by the application of *ksāra anjana* (alkaline caustic ointment). He rightly advised the application of the caustic ointment with an iron rod.

To make *kśāra anjana,* as described by Suśruta, a sufficient quantity of blue barley (with bristles) should be soaked for a week or two in the milk of a black cow and then dried; the milk-soaked barley should then be burned to ashes. These ashes should be mixed with equal parts of the ashes prepared by burning *tūlaśi* (Ocimum album), *kānchana* (Bauhinia variegata), *kapittha* (Feronia elephantum), *vilwa* (Aegle marmelos), *misindā* (Vitex trifolia) and *jati* flowers (Jasminum grandiflorum) and an alkaline solution then prepared by mixing the two ashes with water. This alkaline solution should again be mixed with equal parts of sodium chloride, *tuttha* (cupric sulfate) and *rōchana* (Mallotus philippinensis) and then boiled. The compound thus chemically prepared is most efficacious in the treatment of *balāsa.* Other alkaline preparations may be similarly made with white *barvari* (Ocimum gratissimum), *sūrasā* (Ocimum album), *phanijhaka* (Origanum marjorana), *sūgandhā* (Acorus calamus) and *kapittha* (wood apple, Feronia elephantum) and employed as a caustic *anjana.*

Suśruta's ocular therapeutics for *pistaka* (a grayish looking fleshy spot on the sclera) consists of the application of an *anjana* prepared from a compound of equal parts of *sūnthi* (dry ginger), *pippali* (Piper longum), mūsta (Cyprus rotundus), sodium chloride and seeds of Moringa pterygosperma made into a paste with the juice of *matulūnga* (Citrus limmeta). This is said to be highly efficacious in eradicating the disease.

Of other *anjanas* used for the treatment of this disease, one is made as follows: *Kantakari* (Solanum xanthocarpum) fruits are collected when ripe and the seeds taken out; then a paste made of equal parts of *pippali* (Piper longum) and *srotānjana* (antimony sulfide) is placed inside these seedless fruits for seven nights. The medicament thus prepared is taken out and applied to the eye as an *anjana.* In a similar fashion, inspissated decoctions or pastes of *pippali* and *srotānjana* preserved inside the fruits of *sajinā* (Moringa pterygosperma), *indravārūni* (Citrullas colocynthis) and *patōla* (Trichosanthes dioica) and *amalaki* (Embelica officinalis) may be used as an *anjana.*

Praklinna-vartma (acute gangrenous blepharitis) and *parklinna-vartma* (xerosis conjunctivae), according to the teachings of Suśruta,[87] may be efficaciously treated with an *anjana* prepared from a compound of *kasisa* (ferrous sulfate), *samudraphena* (Sepia officinalis), *rasānjana* (extract of Berberis asiatica) and buds of *jati* (Jasminum grandiflorum). The local itching may be alleviated immediately by the application of an ointment prepared by rubbing equal parts of sodium chloride, seeds of *sajinā* (Moringa pterygosperma) and *manahśilā* (realgar) with the expressed juice of lemon.

Chemosis of the eyelids and conjunctiva, as well as itching, may be relieved by the application of an *anjana* made from equal parts of *sūnthi* (dry ginger), *devadāru* (Pinus deodara), *musta* (Cyprus rotundus), *saindhava* (sodium chloride) and buds of *jati* made into a paste with a sufficient quantity of wine *(sūrā)*.

The application of oleaginous salves plays the main role in the therapy for diseases of the eyelids, preceded by the thorough removal of the crusts and scales with antiseptic and medicated lotions combined with milk, sodium chloride and extract of Berberis asiatica. The physician should regulate the use of the salves according to the urgency of the condition. In addition, energy-giving, soothing drugs and vitamin-containing foods, such as milk, butter, oranges and lemons, are prescribed by Suśruta.

For the treatment of *lagana* (papillary conjunctivitis) and *arso-vartma* (trachoma) the application of copper sulfate and ointments prepared from copper sulfate, piper longum and honey are advised as being efficacious, in addition to surgical measures. Massage of the affected part with medicinal scarifying powders and washing the organ with antiseptic and astringent eye lotions are also advocated by Suśruta.

The medicinal treatment of *raktaja abhiśyanda* and *raktaja adhi-mantha,* (due to blood diseases),[45a] *sirōt-pāta* (pannus), *sirā-harśa* (high grade pannus), *arjuna* (a blood red raised speck on the sclera), *śūkra* (ulcerative and nonulcerative keratitis) and *ajakājāta* (a sarcomatous growth invading the cornea) according to Suśrutian principles are briefly outlined.

The patient with *raktaja abhiśyanda* and *raktaja adhimantha* should be anointed with old and matured clarified butter, and venesection with a *vetasapatra jantra* (*B* of the illustration) should be resorted to. After the bodily toxins have been eliminated by sufficient purging, an errhine composed of clarified butter mixed with sugar should be prescribed for the purification and pacification of the deranged humors of the head. When the system has been cleansed both internally and externally, the physician should prescribe the use of eye washes, medicated plasters, fumigation, the use of medicated snuff and eye lotions and the applica-

tion of *anjanas* and *tarpana* and *snehana* (oleaginous) *putapāka* according to the indications of the disease. *Pralepa* (plaster), a warm medicinal plaster composed of decoctions of equal parts of *nilōtpala* (Nymphaea stellata), *usira* (root of fragrant grass), *daru-haridra* (Berberis asiatica), *kāliya* (white sandalwood), root of glycyrrhiza glabra, *Mūstoka* (Cyprus rotundus), *lōdhra* (Symblocos racemosa) and *padma-kāstha* (Prunus pudum) boiled with washed *ghrta* (clarified butter), should be applied around the affected eye. Mild fomentations (*sveda*) and also venesection by application of leeches are indicated in case there is intolerable neuralgic pain. Suśruta also prescribed large doses of clarified butter as sedative medicament for the alleviation of unbearable pain. Remedial measures described in connection with *pittaja abhiśyanda* should also be prescribed. *Aschyōtana* (eye lotion), consisting of rain water mixed with pulverized *kaśerūka* (Scripus gressus) and *yastimadhu* (glycyrrhiza), should be squeezed through a sterile lint onto the affected eye. *Ananjana* (ointment), consisting of equal parts of *manjisthā* (Rubia cordifolia), *kantakāri* (Solanum xanthocarpum), *vrhati* (Solanum indicum), *amalaki* (Embelica officinalis), *vilwa* (Aegle Marmelos), *arjūna* (Terminalia arjuna), *gāmbhāri* (Gmelina arboria), *patali* (Stereospermum suaveoleus) and *dhātaki* (Woodfordia floribunda) pulverized and mixed together with honey or with the juice of sugar cane, is extolled by Suśruta as being efficacious in the treatment of ophthalmia and uveitis due to disorders of the blood.

A *varti anjana* (ointment stick) prepared from a compound of equal parts of red sandal wood, *kūmūda* (Nymphaea stellata), *tejapatra* (leaf of Courus cassia), *śilājatū* (asphalt) and *kumkuma* (saffron) with the powders of iron, copper and copper sulfate, lead, bell metal, extract of Berberis asiatica and the resinous exudation of a *nimba* (Melia azadirachta) tree and thoroughly mixed with honey possesses scarifying properties and because of its constituent medicinal ingredients is highly efficacious in *adhimantha* (uveitis) as well as in *abhiśyanda* (ophthalmia) caused by disorders of the blood.

Suśruta considered compound of *rasanjana* (extract of Berberis asiatica), honey and clarified butter as a specific cure for *sirōt-pāta* and *sirā-harśa*.[45a] He also regarded as efficacious an *anjana* prepared from equal parts of powders of conch shell, realgar, cupric sulfate, *darū-haridra* (Berberis asiatica) and *saindhava* (sodium chloride) mixed with honey. The other *anjanas* used in the treatment of *sirōt-pāta* are: an ointment prepared from a paste of sodium chloride, *kāsisa* (ferrous sulfate) and breast milk; one made from *swarna-gairika* (red ocher) mixed with honey, and one prepared from white *maricha* (pepper) mixed with the juice of the flowers of *sirisa* (Acacia Lebbec) and with honey and wine (*sūrā*).

For the treatment of *sirāharśa* (high grade pannus), Suśruta prescribed an *anjana* prepared by mixing *rasānjana* (extract of Berberis asiatica) with honey, or by rubbing sodium chloride and ferrous sulfate with honey or by mixing *phānita* (treacle boiled down to a sticky consistence) with honey or *phānita* and *amlavetasa* (sorrel) with breast milk. All these ointments are, according to Suśruta, very efficacious.

In the medicinal treatment of *arjuna* (a blood red, raised speck on the sclera) all the remedial measures described in connection with the treatment of *pittaja abhiśyanda* should be employed, in accordance with the discretion of the attending physician. Eye washes composed of sugar cane juice, honey, sugar, breast milk, *dārū-haridrā* (Berberis asiatica), *yastimadhu* (glycyrrhiza) and sodium chloride boiled in water should be prescribed, or an *anjana* prepared from a combination of the foregoing drugs may be used with advantage. Eye lotions prepared from the decoction of the following substances, namely, sugar, glycyrrhiza, honey, sodium chloride, *vijapāraka* (Citrus limmeta), and *dadima* (Punica granatum) and acid fruits, taken singly or in combinations of two or three each, may be used judiciously.

Anjanas which are specific for *arjuna* are prepared as follows:[45a] by mixing equal parts of *sphatika* (crystal), *pravāla* (pearl), *samkha chūrna* (powders of conch shell) and *yastimadhu* (glycyrrhiza) with honey; by mixing *samkachurna* and *samudraphena* (Sepia officinalis) and sugar with honey; by mixing equal parts of sodium chloride, *nirmaliphala* (Strychnos potatorum) with honey; by mixing *rasānjana* (extract of Berberis asiatica) with honey, or by mixing *kāsisa* (ferrous sulfate) with honey.

The ophthalmic surgeon should also try the *lekhana anjana* (scarifying ointment) in cases of resistant disease. The composition of the *lekhana anjana* is as follows: All kinds of pulverized metals (gold, silver, copper, tin, lead and iron) and metallic substances (realgar and red ocher), sodium chloride, all kinds of pulverized jewels (lapis lazuli, pearl and ruby), the teeth of cows and horses, the horns of cows and horses, pulverized shells of hen's eggs, *lasūna* (garlic), *trikatu* (red pepper, ginger and black pepper), *karanja* (Pongamia glabra) seeds, drugs of the sedative and astringent groups, namely, *kāsisa* (ferrous sulfate) and *elā* (large cardamom seeds), are macerated and mixed with honey, clarified butter and milk.

Suśruta gave the indications for the use of the *lekhana anjana* (scarifying anjana) as follows: After all remedial measures specific for the treatment of *abhiśyanda* (ophthalmia), beginning with venesection and ending with *putapāka,* have been administered, the surgeon should think of the advisability of using *lekhana anjana.*

Next I shall consider the treatment of keratitis (*śukra*) as taught and practiced by Suśruta.[45] He divided keratitis into two types, namely,

savrana-śukra (ulcerative keratitis) and *avrana-śukra* (nonulcerative keratitis). However, before discussing the treatment of keratitis, it should be instructive to consider briefly Suśruta's teachings on the anatomic structure of the cornea, as well as on the pathogenesis and the pathologic changes of corneal inflammation (keratitis).

Suśruta not only knew the macroscopic anatomic structure of the cornea but distinctly mentioned the existence of the different microscopic layers, namely, the superficial layer, the second layer and the deeper layers, which are now known as the substantia propria, Descemet's membrane and the inner epithelial layer. Suśruta's second layer of the corneal substance is now known as Bowman's membrane. This should be more appropriately called the Suśruta-Bowman membrane, since Suśruta first described it in the prehistoric age and Bowman redescribed it in 1849.

As is clear from his writings on the pathology of keratitis in general, Suśruta taught that in ordinary inflammation of the cornea the inflammatory focus is confined to the most superficial layer (*ekatwaggata*); when, owing to prompt and proper treatment, as well as to the local resistance of the corneal tissues, the reparative process sets in, the inflammation does not extend to the second layer and healing occurs, the affected part of the cornea regaining its normal luster and transparency. If, on the other hand, the inflammatory process is stronger and more virulent, it penetrates deeper, invading and involving the second layer (Bowman's membrane) and the deeper layers of the cornea.

The ulcerative types of keratitis (*savrana-śukra*), according to Suśruta, are characterized by the formation of corneal ulcers following the necrosis and suppuration of the inflamed area, as well as by circumcorneal injection and hot exudation, the subjective symptoms being pricking pain, photophobia, lacrimation, spasms of the lids and cloudiness of vision. The distinguishing features of a nonulcerative type of keratitis (*avrana-śukra*) are cellular infiltration of the corneal tissue, involving the superficial as well as the deeper layers, without leading to ulceration, and followed by vascularization of the cornea, which generally simulates a speck of translucent cloud in the sky and is attended with lacrimation, pain and diminution of visual acuity, depending on the severity of the inflammatory process.

Suśruta gave the prognosis of the corneal ulcers as follows: If the ulcer is simple and superficial, it is generally circumscribed by the inflammatory process and clears up uneventfully in the course of a few days, the prognosis being good. Again when the ulcer is not situated too near the pupillary region (center) of the cornea, does not extend deeper than the superficial layer and is not attended with excruciating and pricking pain and continuous hot inflammatory exudation, the prognosis is equally favorable. Once the second layer of the cornea is invaded by the inflam-

matory process and destroyed, as is the case in all varieties of deep-seated corneal ulcers, it is never fully regenerated, and inevitably some degree of permanent opacity of the cornea remains, thus impairing greatly the visual acuity of the organ.

Suśruta considered the prognosis of hypopyon ulcers as always doubtful and grave and the cure as difficult.

Cure may be effected with the greatest difficulty in a case of *avrana-śukra* (nonulcerative keratitis) which is of long standing and accompanied by thickening of the cornea due to cellular infiltration. In such a case if the inflammation is of prolonged duration and the cornea is covered wtih shreds of highly vascularized conjunctival tissue stretching down to the second layer and obstructing vision (interstitial keratitis), cure is also difficult. These observations of Suśruta are in accordance with the findings of Bowman as well as of the modern ophthalmic surgeons—Fuchs, Parsons and others.

Suśruta subclassified keratitis according to the location of the inflammatory focus, as (1) keratitis involving the superficial layer (*ekatwaggata*), (2) keratitis involving the second layer (*dwitwaggata*) and (3) keratitis involving the deeper layers (*gambhirajāta*).

Clinical forms of ulcerative keratitis recognized by Suśruta are simple, compound (*yūgma*) and hypopyon ulcers; those of nonulcerative keratitis consist of raised superficial keratitis (*uttāna-śukra*), herpes corneae (*sankhendu-śukra*), keratitis punctata (*kūnda*), keratitis fasciculata or thickened keratitis (*vahala-śukra*), deep-seated or interstitial keratitis (*gambhirajāta*), keratomalacia (*vaihāyaśābhra-śukra*), rough or sclerosing keratitis (*karkasa-śukra*) and keratitis involving the second layer (*avagādha*).

In connection with the etiology, Suśruta taught that the predisposing and exciting factors of keratitis are: *abhiśyanda* (ophthalmia) in general, trauma to ocular structures, malnutrition of the cornea due to deficiencies in the diet as well as to exposure to dust, smoke, heat and sun, and ocular sepsis due to constant trickling of sweat.

General measures of ocular therapy to be adopted in keratitis are: the use of warm compresses for combating the inflammation; the use of astringent, antiseptic and antiphlogistic eye washes and eye lotions, the administration of sedative medicaments for relieving pain as well as chemosis; cauterization of the ulcerated area in order to limit its spread; application of *rōpana* (healing) and *lekhana* (scarifying) *anjanas* in ulcerative and nonulcerative varieties of keratitis, and the use of sedative *tarpanas* and *putapākas* as well as the administration of light and nutritious diets.

In the treatment of *avrana-śukra* (nonulcerative keratitis) as well as of certain types of *savrana-śukra* (ulcerative keratitis) attended with vascularization of the corneal tissue, Suśruta was the first to conceive

of the deleterious action of vascularization of the cornea, for he observed that the corneal blood vessels caused the aggravation of cellular infiltration and activated the corneal inflammation, and therefore he rightly advocated that devascularization helped to clear up the corneal inflammation much more rapidly and thus accelerated healing. He effected devascularization, or vascular obliteration, mostly by application of his *lekhana anjana* (scarifying ointment), although in certain cases he also used surgical measures.

Suśruta gave the indications for the use of *lekhana anjana* as follows: After all therapeutic measures mentioned in connection with the treatment of *abhiśyanda* (ophthalmia), beginning with venesection, the use of eye washes, eye lotions and snuffs, the application of astringent and antiseptic ointments and cauterization and ending with the application of *putapāka,* have been used, the surgeon should think of the advisability of trying *lekhana anjana.* Suśruta advised that these foregoing measures be used with discretion in the treatment of *avrana-śukra* (nonulcerative keratitis).

He also advised the efficacy of the use of *lekhana anjana* for those types of ulcerative keratitis which are attended with vascularization of corneal and circumcorneal tissue, for he clearly stated that whether the corneal ulcer is superficially situated (*uttāna,* involving the first layer of cornea) or is invading the second layer (*avagādha*) the surgeon should treat the ulcer as well as the corneal and pericorneal vascularization.

Other *anjanas* prescribed by Suśruta in the routine treatment of keratitis (*śukra*) are: an *anjana* prepared from a compound of pulverized copper, powders of *samkha* (conch shell), *manahśilā* (realgar), *maricha* (black pepper) and sodium chloride, each succeeding drug being double the strength of the preceding one, and mixed with honey; an *anjana* prepared from equal parts of powders of *samkha,* stones of *badari* fruits (Tizyphus jujuba), *kataka* (Strychnos potatorum), *drāksā* (grape) and *yastimadhu* (glycyrrhiza), all pounded together and mixed with honey; an *anjana* made of powdered teeth of elephants or horses, *samudraphena* (Sepia officinalis) and flowers of *sirisa* (Acacia lebbec) mixed with honey; one consisting of *samkha, sukti* (oyster shell), grape, *yastimadhu* and *kataka* pulverized in equal parts and mixed with honey; and one prepared from the inner pulps of the stones of beleric myrobalan (*bibhitaka*-Terminalia belerica) mixed with honey. An eye salve made from fried huskless *mudga* pulse (Phaseolus mungo), pulverized *samkha,* and sugar mixed with honey or one prepared by mixing dried extract of glycyrrhiza with honey may be used with advantage.

Suśruta recommended the application of the caustic alkaline ointment (*ksāra anjana*) in the treatment of corneal ulcers. In case of corneal ulcers involving the second layer and attended with pinching and

excruciating pain, he recommended the administration of a sedative *tarpana*. He also advised massaging (*pratisārana*) the corneal ulcer with a compound of equal parts of *maricha* (black pepper), *pippali* (Piper longum), seeds of *śiriśa* (Acacia lebbec) and sodium chloride, applied with an applicator or with *saindhava* (sodium chloride) alone. Massage with these lightly irritating and antiseptic agents furnishes an efficient mode of disinfecting and cauterizing the corneal ulcers.

Suśruta's treatment of leukomas and opacities of the cornea is as follows: An alkaline solution is prepared by dissolving in water the ashes obtained by burning young bamboo sprouts, *arūskara* (Semecarpus anacardium) when ripe, fibers and shell of palmyra palm and coconut palm taken in equal proportions. The burnt ashes of elephants' bones are soaked in the foregoing alkaline solution up to the saturation point. The saturated solution thus obtained is dried up and pulverized and made into an *anjana* with honey. The application of this alkaline *anjana* with an applicator will remove the whiteness of the cornea. After each application, the eye is washed with an astringent decoction of *triphala* (Terminalia chebula, Terminalia belerica and Embelica officinalis).

The treatment of *akśipāka* (suppuration of the globe; sympathetic ophthalmia) is given by Suśruta as follows: The patient's system should be cleansed both internally and externally before application of the local remedies. After fomentations have been applied, the surgeon should resort to venesection, followed by the administration of the other therapeutic measures, namely, eye washes, eye lotions, errhines and *putapāka*, according to the indications in the case.

The following *anjana* is prescribed by Suśruta for application in *aksipāka:* [45a] sodium and clarified butter mixed with an extract of meat juice in a copper vessel and made into an ointment by mixing the same with wine or curd or curd cream. This is said to be efficacious in *aksipāka* with or without swelling. Alternative *anjanas* are made by mixing *ghrta* (clarified butter) with the rust of bell metal in a copper vessel or by mixing breast milk with sodium chloride in a copper vessel; by mixing equal parts of red ocher (*swarnagairika*) and extract of glycyrrhiza with honey or by mixing clarified butter, pulverized copper and sodium chloride with breast milk.

Liquid extract of meat and sodium chloride soaked in clarified butter and mixed with dry ginger (*sūnthi*) and breast milk should be applied to the affected organ as an eye lotion, or an *anjana* consisting of *jāti* flowers (Jasminum grandiflorum), sodium chloride, *sūnthi* (dry ginger), *pippali* (Piper longum) and extract of *vidanga* seeds (Embelia ribes) mixed with honey may as well be profitably applied.

Suśruta recorded the treatment of *puyālasa* (dacryocystitis) as follows: After the application of oleaginous medicament and fomentation, bloodletting and poulticing (*upanāha*), antiseptic irrigations and the

application of *anjanas* are to be employed by the surgeon. An eye salve compounded from *kāsisa* (ferrous sulfate), sodium chloride, liquid extract of ginger or from a combination of all these with copper and iron powders mixed with honey may be used with advantage.

Ajakājālta (a sarcomatous growth invading the cornea) is treated by Suśruta as follows: The slimy watery matter accumulated in the growth is drained by puncturing the sides of the growth with a needle. The incidental wound is stuffed with pulverized beef mixed with clarified butter. In case the lips of the wound are raised, scarification with a *lekhana jantra* (scarifying instrument) may be resorted to as often as is necessary.

Treatment of *praklinna-vartma* (acute gangrenous blepharitis) and *pariklinna-vartma* (xerosis conjunctivae) caused by disorders of the blood is as follows: [45a] The deranged humors are pacified by the application of *sneha* (oleaginous medicament), by venesection, emetics and purgatives and errhines and enemas. After the organ is soothed by the administration of *tarpana,* the physician should resort to the use of eye washes, eye lotions, snuffs and fumigation with antiseptic and sedative drugs, according to the urgency of the condition. A light stimulating and nutritious diet of milk, barley and vitamin-containing fruits is to be given to the patient.

The following *anjanas* are prescribed by Suśruta: an *anjana* prepared by mixing *rasānjana* (extract of Berberis asiatica) with honey, which is highly useful, and one chemically prepared by boiling the decoctions of the leaves and fruits of *amalaka* (Embolica officinalis) in a copper vessel or by boiling the decoction of *triphala* (Terminalia chebula, Terminalia belerica and Embilica officinalis) or *palāśa* (Butea frondosa) or the decoction of *apāmārga* (Achyranthis aspera).

An *aschyōtana* (eye lotion) prepared from equal parts of *mūsta* (Cyprus rotundus), *haridrā* (Curcuma longa), *yastimadhu* (glycyrrhiza), *priyangū* (Aglaia roxburghiana), *sarsapa* (Brassica comprestris), *lodhra* (Symblocos racemosa) and *anantamūla* (Indian sarsaparilla) and boiled either in rain water or in distilled water may be profitably used in *praklinna-vartma* and *pariklinna-vartma.*

A *chūrna* (powder) *anjana* prepared from equal parts of *samudraphena* (Sepia officinalis), sodium chloride, powders of *samkha* (conch shell), *mūdga* (Phaseolus mungo) and the seeds of *sobhānjana* (Moringa pterygosperma) was prescribed by Suśruta to alleviate the itching and also to cure *pariklinna-vartma.* He also used equal parts of *tūttha* (cupric sulfate) and *kajjala* (collyrium prepared from the lampblack collected by burning a wick soaked in castor oil) mixed with honey and applied as an *anjana* in these ocular diseases.

It would be instructive to consider the treatment of *kūkūnaka* [88] (conjunctivitis vernalis; spring catarrh) which Suśruta described in the appendix to his discourses on "Eye Diseases and Their Treatment." Probably owing to the uncommonness of its occurrence, it has been treated in the appendix. Suśruta clearly taught that the other ocular diseases which he described in the text occur in young as well as in elderly persons, whereas the ocular disease known as *kūkūnaka* occurs chiefly in children. According to him, it is a chronic inflammatory disease affecting the *netra-vartma* (eyelid) of boys, the subjective symptoms being excessive itching, photophobia and marked lacrimation. The symptom which annoys the patient most is the intolerable itching, so much so that boys affected by this disease can be seen to rub their eyes as well as the nasal and the supraorbital regions of the eyes. According to Suśruta, the tide of this disease ebbs or flows according to the coolness or heat of the atmospheric condition; that is to say, the disease becomes aggravated in the summer and lessens or disappears in the winter. Suśruta most excellently depicts the most outstanding symptom of this disease when he says that the patient does not in the least tolerate *sūryaprabhā* (the sun's rays). These scientifically accurate observations of Suśruta on the characteristic and predominant features of *kūkūnaka* are much akin to those of Arlt and Saemisch. Regarding the etiology, Suśruta attributed the incidence of this disease to bodily toxins from dietetic disorders resulting in the derangement of the bodily humors and excited and aggravated by sunlight (*sūryaprabhā*). Like Dimmer and Kreibich, Suśruta incriminated sunlight as the causative and exciting factor.

As for the therapeutic measures, Suśruta advocated the elimination of bodily toxins by means of emetics and purgatives. He also advised venesection by the application of leeches, as well as scarification (*lekhana*) of the eyelid with a *śephalikā* leaf (Nyctanthes arbortristis) or a *lekhana sastra* (scarifying instrument). Massage of the affected eyelid (*pratisārana*) with a medicinal compound prepared by mixing pulverized *trikatu* (Piper longum, dry ginger and black pepper) with honey is also recommended. The eyes of the patient must be duly protected from sunlight and heat by bandaging them and keeping the patient in a chamber unexposed to these elements. The inflammation is combated by irrigating (*praksālana*) the eyelid with mild astringent eye washes (*pariśeka*) prepared from the decoction of *amalaka* (Embelica officinalis) and the leaves of *asmantaka* (wood sorrel, Rumex scutatus), *āmra* (Mangifera indica) and *jambu* (black plum—Eugenia jambolana). An eye lotion prepared by cooking clarified butter with *gūlancha* (Tinospora cordifolia) or with *triphala* (myrobalan, Terminalia belerica and Embelica officinalis) is also recommended.

88. Suśruta Saṁhita,[1] Uttara Tantra, chap. 19, appendix.

The following *anjanas* are prescribed by Suśruta for application in spring catarrh: an *anjana* prepared by mixing *manahsilā* (realgar), *maricha* (black pepper), *samkha* (conch shell) and *rasānjana* (extract of Berberis asiatica) with sodium chloride, molasses and honey; or by mixing *trikatu* (Piper longum, dry ginger and black pepper), *palāndū* (onion), *yastimadhu* (glycyrrhiza), sodium chloride and shellac (*lākśa*) and red ocher (*gairika*) with honey; or by mixing leaves of *nimba* (Melia azadirachta), glycyrrhiza, *haridra* (Curcuma longa) and *darū-haridrā* (Berberis asiatica), powdered copper and *lōdhra* (Symblocos racemosa) with honey, all the drugs being used in equal parts. The application of these *anjanas* should speedily alleviate the itching and bring about amelioration of the disease.

From Suśruta's teachings one learns that night blindness (*kapha-vidagdhadrusti*) occurs from nutritional disorders and avitaminosis and that it can be cured mainly by strengthening diets and by the internal administration of vitamin-containing drugs, such as medicated *ghrta* (clarified butter) and cooked liver and liver extract.[25] Interpreted in the light of modern science, Suśruta's teachings on night blindness mean that the disease is due to a lowering of the nutritional state, i. e., avitaminosis A, and that the eating of cooked liver, which is the storehouse of vitamin A, would expedite the cure of this ocular disease. The prognosis of night blindness, according to Suśruta, is favorable, as it is easily amenable to treatment.

Regarding its treatment, Suśruta laid great stress on ameliorating the general nutrition of the patient by giving vitamin-containing foodstuffs and drugs. Besides this, he advocated local application of ointments containing vitamins, i. e., an ointment prepared from liver extract and butter or clarified butter. He prescribed the internal medication of clarified butter cooked with *pattikālōdhra* (red variety of Symblocos racemosa) or simply old clarified butter. Consumption of milk, vitamin-containing fruits and edible vegetables as well as the taking of *traibrt ghrta* (clarified butter cooked with *tribrt*—Iponioca turpethum) is recommended. Among the edible vegetables, *agastipūspa* (Agati grandiflora), *pālankyā* (spinach) and *sunisannaka* (Blepharis edulis), and among the animal foods, meat of deer and fowl, as well as fish, are prescribed. Attention is also to be directed toward the cleansing of the system both internally and externally. As for the consumption of liver, Suśruta advised that the patient should be given the liver of a goat or sheep cut into pieces, mixed with clarified butter and roasted in fire. Cooked liver is also given along with honey.

The liver of a *gōdhā* (a variety of wild lizard) should be partially opened and packed with powdered *pippali* (Piper longum) and roasted in fire. The application of this compound as an *anjana* should at once cure night blindness. Similarly, an ointment prepared from the liver

of a goat stuffed with *pippali* (Piper longum) and roasted in fire should also effect a speedy and sure cure on one application.

Alternative medicaments prescribed by Suśruta are prepared as follows: Both the spleen and the liver of a goat or a lizard should be chopped into pieces, mixed with clarified butter and roasted slightly. Used internally with honey or as an *anjana,* this medicament is sure to cure an attack of night blindness.

The fat and liver of a goat with a quantity of clarified butter skimmed out of the milk of a she-goat should be mixed with *pippali* (Piper longum) and sodium chloride and a chemical preparation made by boiling these ingredients with the expressed juice of *amalaka* (Embelica officinalis). The preparation thus obtained should be mixed with honey and carefully kept in a closed vessel made of *khadira* (catechu) wood. The use of this chemically prepared compound as an *anjana* is highly recommended for the treatment of night blindness.

Again, *harenū* (Piper auranticum), *pippali* seeds (Piper longum), *elā* (large cardamom seeds) and the liver of a goat or sheep may be pounded together and made into an *anjana* which will be equally efficacious in night blindness.

Suśruta also considered the application of a *varti anjana* (ointment stick) prepared from equal parts of *srotānjana* (antimony sulfide), *saindhava* (sodium chloride), *pippali* (Piper longum) and *renūkā* (Piper auranticum) and mixed with the urine of a she-goat to be highly efficacious. Similar applications of *varti anjana* prepared from equal parts of tagara (Valeriana hardwicitrii), *pippali* (Piper longum), *sūnthi* (dry ginger), *yastimadhu* (glycyrrhiza), *tālisapatra* (Pinus webbiana), *haridrā* (Curcuma longa), *dārū-haridrā* (Berberis asiatica) and *mūsta* (Cyprus rotundus), pounded together and mixed with the liquid extract of cow dung, are also recommended.

For the treatment of *pitta-vidagdhadrusti* (day blindness) Suśruta advised the use of a *gūtikā* (tablet) *anjana* prepared from equal parts of soduim chloride, *srotānjana* (antimony sulfide), *manahsilā* (realgar), *haridrā* and *dārū-haridrā, raktachandana* (red sandalwood, *maricha* (Piper nigrum) and *simbi* (Dolichos lablab), pounded and mixed with the liquid extract of goats' or sheep's liver. The prognosis of day blindness, according to Suśruta, is good, the disease being amenable to treatment.

Suśruta's treatment of *timira* (retinitis) in general consists in the administration of medicaments for eradicating the cause of the disease as well as the prescribing of vitamin-containing and energy-containing foodstuffs for improving the general health.[25]

As it is impossible in this short monograph to go into details of treatment, I shall conclude the outline of ocular therapeutics with a brief narration of Suśruta's advice in regard to diet therapy for retinitis (*timira*) in general.

Suśruta said that a person who with care partakes of old ghrta (clarified butter), *triphala* (Terminalia chebula, Terminalia bellerica and Embelica officinalis), *śatāvari* (Asparagus racemosis), *patōla* (Trichosanthes dioica), *mūdga* (Phaseolus mungo) *amalaka* (Embelica officinalis) and barley in his general dietary has little to fear from an attack of even a dreadful type of *timira*. A *pāyasa* (porridge) prepared simply from *śatāvari* or with *amalaka* or a meal of barley prepared with the decoction of *triphala* and a sufficient quantity of clarified butter should be taken with benefit in cases of *timira*. The partaking of such edible vegetables as *jivanti* (Celtis orientalis), *sūnisannaka* (Blepharis edulis), *vāstūka* (Chenopodium album), *tandūliyam* (Amarantus spinosus), chilli (a variety of red *vāstūka*) and *mūlaka* (Raphanus sativus—radish), as well as of the meat of birds, such as fowl, and of wild animals, such as deer, should be considered as invigorating to the sight. Also, the use of leaves or fruits of *patōla* (Trichosanthes dioica), *karkotaka* (Momordica cochinchinensis), *kāravella* (Momordica charantia), *bārtāku* (brinjal, *tarkāri* (Premna seratifolia), *karira* (tender bamboo plants) and leaves of *sobhānjana* (Moringa pterygosperma) cooked with clarified butter should also prove highly beneficial and invigorating.

COMMENT

An analytic, critical and comparative study of ocular therapeutics as practiced and taught by Suśruta in the prehistoric age has been focused on modern ophthalmic science. A judicious, scrutinizing and unprejudiced review of Suśruta's ophthalmic works should at once convince the most persistent critic that the Suśrutian teachings on ocular therapy are original and exhaustive and, what is more remarkable, that the principles of Suśrutian ocular therapy are mostly scientific. The array of metallic and nonmetallic ocular medicaments which are in universal use in ophthalmic practice today and which are among the main weapons in the therapeutic armamentarium of the ophthalmic surgeon were first introduced into the field of ophthalmology by Suśruta. Among the well known metallic ocular medicaments may be mentioned: gold, which is highly extolled by Suśruta for its tonic, antiseptic, sedative and invigorating properties and as being highly beneficial to the eye; silver and its salts, for their astringent, bactericidal and demulcent and reparative properties, and copper and copper sulfate, for their antiseptic, astringent and scarifying (*lekhana*) properties. Besides this, iron and iron sulfate, zinc, mercury, tin, bell metal, sodium chloride and boric acid are among the other therapeutic agents employed in ocular practice. The therapeutic efficacy of red coral (*pravāla*), pearl (*muktā*), ruby, lapis lazuli, sapphire, diamond (*hiraka*) and crystal (*sphatika*) was known to Suśruta, who used them in ocular therapeutics for their anti-

septic, sedative and scarifying properties and because they were extraordinarily useful as invigorating ocular medicaments.[89] In addition to these, Suśruta was the first to use *darū-haridrā* (Berberis asiatica), *rasānjana* (extract of Berberis asiatica), *triphala* (Terminalia chebula, Terminalia belerica and Embelica officinalis), *srotānjana* (antimony sulfide, marine oyster shell, *samudraphena* (Sepia officinalis), *samkha* (conchshell), *tuvaraka* (Symblocos racemosa) and myriads of other drugs culled from the vegetable, mineral and animal kingdoms, the experimental study of which would greatly benefit the modern ophthalmologist.

Suśruta will ever be remembered by the ophthalmic profession for having first discovered the therapeutic efficacy of copper sulfate (*tuttha*), which has been a great boon to patients suffering from trachoma (*arsovartma*) and papillary conjunctivitis (*lagana*) as well as allied diseases of the eye. In connection with the use of copper sulfate, Gifford [58] writes: "Copper sulfate has been used for thousands of years and is still the drug which is apparently of most value in trachoma." Indeed, it was Suśruta who first tried this drug in the treatment of papillary conjunctivitis as well as of trachoma.

Suśruta used gold as an important constituent of *anjanas* (ointments) in the treatment of *abhiśyanda* (ophthalmia) and of inflammatory diseases of the eyelid, as well as of *adhimantha* (uveitis).[90] Suśruta's teaching on the efficacy of gold salts in ocular therapy is being revived, and in the present century such treatment has been given an extensive trial by Wilson (1928), of Cairo, with relatively good results.[91]

It was Suśruta who pioneered in the introduction of vitamin therapy in ocular practice.[92] His methods of combating the ocular diseases in general by means of antiseptic and antiphlogistic eye washes, eye lotions, compresses, medicated plasters (*pralepa*) and ointments (*anjanas*), as well as by venesection and cauterization, are exactly like those of the modern ophthalmologist. His teachings on the anatomic structure of the cornea and on the causes, pathologic appearance, prognosis and classification of keratitis (*śukra*) are in accordance with the teachings of Bowman [44] as well as with those of the modern authorities. His masterly observations on the causes, symptoms and prognosis of spring catarrh [88] (*kūkūnaka*), which are in agreement with those of the medieval as well as with those of the modern authorities, namely, Arlt,[93] Saemisch,[93] Dimmer and Kreibich [93] and Fuchs and others, show the

89. Suśruta Saṁhitā,[1] Sūtrasthāna, chap. 46.

90. Suśruta Saṁhitā,[86] verse 5.

91. Wilson, R. P.: Rep. Mem. Hosp., Giza **3**:74 and 78, 1928; Ophthalmia Aegyptiaca, Am. J. Ophth. **15**:397, 1932.

92. Suśruta Saṁhitā (footnotes 63 and 85, verse 22).

93. Cited by Fuchs,[83] p. 106.

extraordinary development of his scientific knowledge. Living in an age far removed from the facilities afforded by modern scientific appliances, Suśruta's contributions to ophthalmic science are indeed varied and comprehensive, original and unique and withal most scientific and unparalleled in the history of medicine.

I shall now attempt to review briefly the teachings of Suśruta on ocular therapeutics and, to the best of my capacity, to interpret and evaluate them in the light of modern ophthalmic science.

It will come as a surprise to many, as it did to me, that vitamins were first discovered in the prehistoric age and that the introduction of vitamin therapy into ocular therapeutics was first made by Suśruta.[63] As is clear from his writings, Suśruta recognized the existence and the importance of the vitamins and their efficacy in ocular therapeutics. His masterly exposition on the existence and efficacy of the essential food factors, which he so appropriately called *hrdya* (energizing to the heart) and *jibaniya* (life-giving), and which came to be known as vitamins after their rediscovery by Hopkins[94] and Funk,[95] and his advocacy of vitamin therapy for various ocular diseases heralded an entirely new era in the history of the evolution of ocular therapeutics.

Suśruta prescribed vitamins in the form of milk, butter, cream, old *ghrta* (clarified butter), medicated milk, sugar cane juice, liver and liver extract, lemons and oranges. From his advocacy of vitamin-containing foodstuffs and medicaments, it is clear that he knew that certain ocular diseases, such as night blindness and xerosis conjunctivae, to name only a few, are due to deficiency of vitamins.

Suśruta's teaching in regard to the therapeutics of night blindness[25] is so scientific and elaborate that the modern ophthalmologist could adopt it with the greatest advantage. He prescribed vitamin therapy in the form of cooked liver and liver extract mixed with honey, in addition to advising the use of butter, *ghrta* (clarified butter) and green vegetables, such as *agastipuspa* (Agati grandiflora).

In tracing the evolution of ocular therapeutics with regard to vitamin therapy, one can see how Suśruta's teachings on liver therapy (i. e., vitamin A therapy) in night blindness were followed by Hippocrates as well as by the medical authorities of the post-Suśrutian period.

Hippocrates[2] prescribed raw ox liver with honey for the treatment of night blindness. In Japan and China extracts of hen's liver and the liver of sheep and hen were much applied in olden days.[96] Baizeau[13]

94. Hopkins, G., cited by Tidy, H. L.: Synopsis of Medicine, ed. 4, Bristol, John Wright & Sons, Ltd., 1925, p. 343.

95. Funk, C.: The Vitamines, translated by H. E. Dubin, Baltimore, Williams & Wilkins Company, 1922.

96. Sen, K.: Night Blindness and Vitamin A Deficiency, Proc. All-India Ophth. Soc. **4**:77, 1935.

(1861), an eminent surgeon belonging to the French army, noticed the incidence of epidemic night blindness in soldiers and successfully treated it with the internal administration of cod liver oil. Bitot [14] (1863), another eminent ophthalmic surgeon, recorded 29 cases of xerosis conjunctivae occurring in association with night blindness in older children in a foundling hospital in Bordeaux, the disease being cured with liver therapy. Many Russian investigators have also observed the incidence of night blindness, xerosis conjunctivae and other diseases, during the long Lenten fasts in which all animal foods, including milk and butter, were scrupulously withheld. Blessig [13] (1866), a well known ophthalmic surgeon of the nineteenth century, whose name is associated with Blessig's cavities, observed many such cases of night blindness and xerosis conjunctivae, and his finding was that the derangement of nutrition attended with avitaminosis A during the period of fasting was the cause.

Coming to the twentieth century, one finds that several scientific investigators, namely, Zak [15] (1917, 1920), Hift [15] (1918), Meisner [15] (1919) and others, noticed the occurrence of night blindness among the Austrian prisoners in Russia during the late World War. These Russian physicians also found that the disease was common among the Russian villagers, the condition being more generalized during the period of Lenten fasting, owing to the compulsory abstinence from all sorts of animal foods. As curative remedies for these diseases, the administration of slightly cooked liver and cod liver oil was efficacious. Mori [15] (1923) observed the incidence of night blindness in Japan among coolies working in rice fields during summer. He also observed the condition among soldiers in active training as well as in railway workers in Manchuria, China. Cures following the administration of cod liver oil have been recorded by him.

Coming to more recent times, one finds that Pillat [16] (1929) recorded the incidence of night blindness due to avitaminosis A in a military camp. He effected cure by the administration of cod liver oil and the use of green vegetables. Aykroyd [17] (1930) made learned observations on the incidence of night blindness in Labrador and Newfoundland. His finding was that the disease was due to deficiency or lack of vitamin A and that cure was achieved by cod liver oil therapy. Spence [18] (1931) also made similar observations in England. Jeans and Zentmire [19] (1934, 1937), Park [20] (1935, 1936), Frandsen [21] (1935) and Jeghers [22] (1937) have demonstrated the existence of vitamin A deficiency attended with night blindness.

The association between avitaminosis A and night blindness which Suśruta observed in the prehistoric age has been experimentally confirmed by Fridericia and Holm.[23] Again the finding of Suśruta that liver is the storehouse of that type of vitamin which is specific for the

cure of night blindness (i. e., vitamin A) is being confirmed and corroborated by the experimental studies of Wolff.[24]

Suśruta not only advocated general vitamin therapy in ocular diseases but he understood the therapeutic efficacy of local application of vitamin-containing medicaments[26] (i. e., medicaments compounded with vitamin A products, namely, butter, clarified butter [*ghrta*], cream, sugar cane juice, milk of cows and goats and breast milk), liver extract ointments, ointments prepared with lemon juice or meat extract mixed with butter. A cursory study of Suśrutian ocular therapeutics should at once bring home to the reader that Suśruta used local vitamin therapy (i. e., vitamin A therapy) in various ocular diseases. To cite a few instances, he prescribed an ointment prepared from liver extract and butter or honey for local application in night blindness, as well as ointments containing vitamins for application in blepharitis, xerosis conjunctivae and allied ocular diseases. His method of washing the eye with medicated milk and his *tarpana* measure of filling the eye with medicated or simple *ghrta* (clarified butter) are based on local vitamin therapy.

The local application of preparations containing vitamins, i. e., vitamin A preparations with butter, clarified butter and liver extract, was thus first introduced by Suśruta into the domain of ocular therapy in the prehistoric age. Thanks to the erudite researches of the Russian observers, Balachovski[27] and others, this Suśrutian principle has been revived in the twentieth century and popularized through the efforts of Frederici,[97] Stevenson,[98] Kaptsiovskaya,[28] Rachevskiy,[99] Grósz[100] and others.

Thus one sees that the learned observations of Suśruta on the efficacy of vitamin therapy, both local and general, in ocular diseases are most scientific and have been upheld by medical scientists all over the world from the prehistoric age to the present time.

In connection with keratitis[45] *(śukra),* one finds that much like the modern authorities, Fuchs,[101] Parsons[102] and others, Suśruta classified keratitis on the clinical basis into ulcerative *(savrana-śukra)* and nonulcerative keratitis *(avrana-śukra).* He further subdivided the dis-

97. Federici, E.: Le vitamine A e D nei processi di riparazione della cornea (studio sperimentale), Boll. d'ocul. **16**:357, 1937.

98. Stevenson, E.: Cod Liver Oil as a Local Treatment for External Affections of the Eyes, Brit. J. Ophth. **20**:416, 1936.

99. Rachevskiy, F. A.: Provitamin A as Anti-Inflammatory Factor in Diseases of the Eye, Sovet. vestnik oftal. **4**:470, 1934.

100. de Grósz, S.: Local Use of Vitamin A Preparations in Ophthalmic Practice, Arch. Ophth. **22**:727 (Nov.) 1939.

101. Fuchs,[33] p. 160.

102. Parsons,[34] p. 194.

ease, according to the location of the inflammatory focus, into keratitis involving the superficial layer of the cornea *(ekatwaggata)*, that involving the second layer *(dwitwaggata)* and that involving the deeper layers of the cornea (gambhirajāta).[45a]

Again like the modern ophthalmologists, Suśruta subclassified non-ulcerative keratitis *(avrana-śukra)* into the following clinical entities: raised superficial keratitis *(uttāna-sukra)*, herpes corneae *(sankhendu-śukra)*, keratitis punctata *(kūnda)*, keratitis fasciculata *(bahala-śukra)*, interstitial keratitis *(gambhirajāta-sukra)*, keratomalacia *(vaihāyasābhra* -[of the color of cloud]*-śukra)*, sclerosing keratitis) *(karkasa-śukra)* and keratitis involving the second layer *(avagādha-śukra)*.[45a]

Suśruta's knowledge of the anatomic structure of the cornea as well as of the pathologic appearance and pathogenesis of corneal inflammation is elaborate and scientifically up-to-date. Indeed, many of the teachings of Bowman [44] in this respect are in agreement with those of Suśruta.

Suśruta knew and clearly described not only the macroscopic anatomic structure of the cornea but the component microscopic layers of the cornea, namely, the superficial layer, the second layer and the deeper layer, which are now known as the substantia propria, Descemet's membrane and the inner epithelial layer. Suśruta's second layer of corneal substance now goes by the name of Bowman's membrane, Bowman having discovered it in 1849. This would be more appropriately styled as the "Śuśruta-Bowman membrane," since Suśruta described it first, in the prehistoric age, and Bowman redescribed it in the nineteenth century.[45a]

Suśruta's teachings on the prognosis of corneal ulcers are as true today as they were in the Suśrutian period.[45a] Suśruta taught that if the corneal ulcer is superficial and simple in nature, the inflammatory process being generally circumscribed, it clears up uneventfully in a few days, the prognosis being favorable. Also, when the ulcer is not situated too near the pupillary region (i. e., near the center of the cornea), does not extend deeper than the superficial layer and is not attended with unbearable pain and hot exudation, the prognosis is equally favorable. If the second layer of the cornea is invaded by the inflammatory process and destroyed, as is the case in all types of deep-seated ulcerative keratitis, it is never fully regenerated, and inevitably some degree of permanent opacity of the cornea remains, thus impairing greatly the visual acuity. The prognosis in such cases is grave. For comparison with the modern prognosis of keratitis ulcerosa, the following quotation from Fuchs [103] is cited: "The prognosis for vision depends upon the location, the extent and the density of the opacity which the ulcer

103. Fuchs,[33] p. 162.

will leave behind. . . . When an ulcer advances toward the center of the cornea, each millimetre thus damages the vision further, whereas the expansion toward the margin of the cornea is pretty much a matter of indifference. Again Suśruta taught that the prognosis of hypopyon ulcer is always doubtful, being grave and difficult of cure."

Fuchs gave the prognosis as follows: "Extensive suppurations of the cornea, as in conjunctivitis gonorrhoica, ulcus serpens, etc., often leave a narrow marginal portion of the cornea unattacked; of course, this is not often adequate for an iridectomy, and the restoration of vision is therefore impossible."

According to Parsons:[104] "An immense amount of research has been devoted to inflammation of the cornea from the earliest investigations of Bowman (1849) onwards, and much of our knowledge of inflammation in general is derived from these researches."

Indeed, most of the findings of Suśruta in connection with his investigations on the anatomic structure of the cornea, as well as the pathologic picture of corneal inflammation, are exactly in accordance with those of Bowman.

The observation that reducing the supply of blood to the cornea has a healing effect on various forms of keratitis (*śukra*) was first made by Suśruta, who well understood the deleterious effects of vascularization of the cornea. He therefore rightly advocated vascular obliteration by means of his *lekhana* (scarifying) *anjana,* in addition to the employment of surgical measures.[45a]

Among the enthusiastic exponents of the Suśrutian principle of vascular obliteration in the treatment of various forms of keratitis may be mentioned such famous persons as Vagbhatta[37] (about 700 A. D.), a Hindu surgeon of India belonging to the Suśrutian school, who performed devascularization by means of *lekhana anjana* as well as by surgical means; Boury[39] (1743), who performed devascularization by cutting the pericorneal blood vessels; de Saint Yves[40] (1767), who effected vascular obliteration by operative measures; Janin[38] (1772), who used mostly an astringent ointment named after him; Scarpa[41] (1801), who advocated vascular occlusion by means of application of astringent ointments as well as by recision, and Bell[42] (1816), who also recommended extirpation of the pericorneal blood vessels by surgical means.

Coming to the premodern period, the names of the famous ophthalmic surgeons who advocated vascular obliteration in the treatment of various types of keratitis, either by syndectomy, peridectomy or peritomy, are

104. Parsons,[34] p. 193.

Bader [105] (1863), Furnari [106] (1862), Lawson [107] (1863-1865), Weller [108] (1826) and Ziegler [109] (1898), while those of the twentieth century are Teale [110] (1901), Whitehead [111] (1922), Alonso [112] (1922), Gallemaerts [113] (1923), Szokolik [114] (1927), D'Amico [115] (1930) and Gundersen [48] (1939), who has made a thorough and learned investigation into the problem.

Indeed, what Vagbhatta, Boury and others conceived in the medieval period and Denig [116] and Gundersen and others conceived in the modern period regarding the deleterious action of vascularization on corneal tissues in various forms of keratitis, Suśruta's master mind conceived in the prehistoric age. Of course, as science has advanced with the progress of time, the technic of effecting devascularization has been modified, as by applications of astringent and scarifying ointments, by surgical measures or by diathermy, the principle remaining the same.

The services of the cautery, both *agni-karma* (thermocautery) and *kṣāra-karma* (caustic cautery), were first employed by Suśruta for the treatment of many of the obstinate ocular diseases. Suśruta advised cauterization in the treatment of corneal ulcers, uveitis *(adhimantha),* trichiasis *(paksma-kopa),* chalazion and various obstinate inflammatory diseases of the eyelid *(vartma roga).*

Rightly did Suśruta observe: "Of all cutting instruments and their substitutes, caustics or vegetable alkalis are the most important, because by means of them deep and superficial incisions and scarifications may be made."

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105. Bader, C.: On Syndectomy, Ophth. Hosp. Rep., London **4**:19, 1863-1865.

106. Furnari, S.: De la tonsure conjonctivale et de son efficacité contre les lésions panniformes et chroniques de la cornée et contre les ulcérations vascularisées et les opacités interlamellaires de cette membrane, Gaz. méd. de Paris **17**:54, 83, 114, 147, 176 and 210, 1862.

107. Lawson, G.: Case of Vascular Cornea and Granular Lids; One Eye Treated by Inoculation, the Other by Peritomy, Ophth. Hosp. Rep., London **4**:64, 1863-1865.

108. Weller, C.: Die Krankheiten des menschlichen Auges, Berlin, Schüppel, 1826, p. 208.

109. Ziegler, S. L.: The Surgery of Trachoma, J. A. M. A. **30**:131 (Jan. 15) 1898.

110. Teale, T. P.: Tr. Ophth. Soc. U. Kingdom **21**:146, 1901.

111. Whitehead, A. L.: Ocular Tuberculosis, Brit. J. Ophth. **6**:529, 1922.

112. Alonso, A. F.: La peritomia en las úlceras crónicas vasculares de la cornea, Tr. Internat. Cong. Ophth. **1**:371, 1922.

113. Gallemaerts: Péritomie por clivage conjonctivale, Ann. d'ocul. **160**:342, 1923.

114. Szokolik, E.: Peridektomie der Bindehaut, Klin. Monatsbl. f. Augenh. **78**:693, 1927.

115. D'Amico, D.: Cong. Soc. ital. di oftal. **10**:22, 1930.

116. Denig, R.: Transplantation bei chronischem trachomatosen Pannus, Ztschr. f. Augenh. **25**:278, 1911.

Suśrutian teachings called attention to the superior efficacy of thermocautery,[10] and bear a striking similarity to those enunciated by Hippocrates.[2] Suśruta's views are also identical with those of the Roman,[79] Greek [83] and Arabic,[4] as well as the Hindu,[8] surgeons of the post-Suśrutian period. Even in the modern period, the views of Coulter,[31] Monbrun and Castéran,[32] Fuchs,[33] Parsons [34] and others in connection with cauterization in ocular practice are almost identical with those of Suśruta.

Suśruta's observations on the causes, symptoms and prognosis of spring catarrh *(kukunoka)* [88] are in accordance with the findings of Arlt and Saemisch,[93] of Kreibich and Dimmer [93] and of Fuchs [117] and others. Suśruta wrote that besides toxins elaborated from dietetic disorders, sunlight *(suryaprabhā)* is the main exciting factor in the causation of the disease, which is much like the theory of Kreibich and Dimmer. And what is more remarkable and significant is that this Suśrutian teaching is supported and corroborated by the recent investigations of Mans, Froge and Chiniara,[118] such a causation being supported by experimental as well as by clinical data.

In connection with the symptoms, Suśruta taught that the tide of the disease ebbs or flows according to the coolness and hotness of the atmospheric condition. This is exactly in accordance with the findings of the medieval as well as of the modern authorities, namely, Arlt, Saemish, Fuchs, Parsons and others.

Suśruta's observation that spring catarrh is an uncommon ocular disease affecting the eyelids and occurring mostly in *bālaka* (boys) is strikingly in agreement with modern findings. In his pen picture of the predominantly characteristic symptoms of the disease, namely, excessive and intolerable itching *(kanduyana)*, photophobia and lacrimation, he is supported by the medieval as well as the modern ophthalmologists. His view that spring catarrh is a curable disease, though recurrent, is similar to that of modern authorities.

As for the treatment of spring catarrh, Suśruta advised venesection in acute exacerbations of the disease, scarification and expression of the eyelid with a *lekhana sastra* (scarifying instrument) or with a *śephālikā leaf* (Nyctanthes arbortristis) as well as protection of the patient from exposure to light. He also recommended washing the eye with mild astringent lotions and applying sedative ointments for the relief of itching. His teachings are elaborate and can be advantageously tried by modern ophthalmologists.

The use of the magnet was known to ancient Hindu surgeons, and in the Suśrutian age it was used to extract foreign bodies from the

117. Fuchs,[33] pp. 105-106.

118. Froge, P., and Chiniara, J.: Sunlight and Vernal Conjunctivitis, abstracted, Arch. Ophth. **22**:924 (Nov.) 1939.

eyes.[65] In the modern period, Haab Sr. and Haab Jr., Mellinger and others reintroduced the magnet for extracting particles of iron from the eye.

Suśruta's ocular materia medica is exhaustive, consisting as it does of metals and metallic salts and drugs culled from the vegetable kingdom, as well as of mineral salts and jewels, which are too numerous to be reviewed in detail. The therapeutic efficacy of mother's milk as a hemostatic, soothing and nutritive medicament was first known to Suśruta. Truly speaking, mother's milk was the epinephrine of the preepinephrine days, and it thus formed a ready weapon in the armamentarium of the ophthalmic surgeon for the controlling of intraocular hemorrhages. A thorough analytic study of the Suśrutian ocular materia medica would greatly benefit the modern ophthalmologist, and might add numerous new medicaments to modern ophthalmic practice.

Among the general measures employed by Suśruta in combating ocular diseases are: irrigation of the eye with antiseptic, antiphlogistic and astringent eye lotions, eye washes and eye drops and the use of compresses, medicated plasters, ointments, fumigation and snuffs. Among the special therapeutic measures, he employed *tarpana*[62] and *putapāka*.[62] His instructions regarding the preparation and use of the different varieties of *anjanas* (ointments) are most elaborate as well as scientific.[62] The distinctions between the different varieties of *anjanas* into *rōpana* (healing), *lekhana* (scarifying), *snehana* (oleaginous) and *prasādana* (invigorating) anjanas containing the special medicinal ingredients characteristic of each, and their appropriate applications in different ways, speak of the highly specialized knowledge and scientific originality and skill displayed by Suśruta. What the modern surgeon's craftsmanship achieved by operative methods, that and perhaps much more was attained by Suśruta's *lekhana anjana*. While the technic of the preparation of *putapāka* would smack of medievalism to the modern ophthalmic surgeon, the principle involved in its use is certainly scientific, and the ingenuity and scientific originality exhibited by Suśruta in the use of *anjanas* (ointments) in ocular practice appear ultramodern. With slight modifications, perhaps, the technic of *putapāka* can be used with benefit in modern ophthalmic therapeutics.

Indeed, the scientifically high standard of teaching ocular therapeutics, as disclosed in the ophthalmic works of Suśruta, and also the scientific accuracy thereof when compared with modern ophthalmic knowledge, are really surprising when one considers the early age in which Suśruta lived.

Nor was Suśruta dogmatic in his teachings. True phophet of science that he was, he frankly confessed that he sowed the seeds of ophthalmic

science, nurtured, with fond care and fatherly benediction, the tender ophthalmic plant and left it to grow luxuriantly to bear good fruits under the congenial heat of the medical genius of the succeeding generations. No wonder, therefore, that the prophetic words of the father of ophthalmic science are being happily fulfilled.

Much as I should have liked to deal exhaustively with Suśruta's teachings on ocular therapeutics, the scope of this paper does not allow me to do so. I have therefore tried to epitomize the principles of Suśrutian teachings on ocular therapy. Judged in the light of modern ophthalmic science, his teachings are scientific as well as exhaustive, and the modern ophthalmologist could study them with advantage and admiration.

In presenting this contribution on the most original and valuable ophthalmic works of Suśruta, I have attempted only to add a hitherto unwritten, but most important, chapter in the history of the evolution of the science of ophthalmology. In the compilation of my paper I have freely consulted the works of numerous world authorities, both ancient and modern.

I have tried, as far as I could, to interpret the teachings of Suśruta on ocular therapeutics and to evaluate them in the light of modern ophthalmic science. Because Suśruta as the world's first ophthalmic surgeon nurtured and fathered the science of ophthalmology in the prehistoric age and as a great scientific genius made contributions to the domain of ophthalmology which are unparalleled in the history of medicine and presented principles of ocular therapy which remain as true today as when he first taught them, his works should ever be acknowledged as the classical lore in medicine and should be profitably explored for the purpose of enlarging the scope and enriching the body of modern ophthalmic science.

A true history of ophthalmology would not be complete without mentioning the name of the pioneer surgeon who, for the first time in the prehistoric age, built ophthalmic science on a rational basis, nor would it be complete without paying homage to those great seers of science, of ancient as well as of modern periods, who by their perseverance and selflessness, unraveled the mysteries and enriched the body of ophthalmic science. Hence, if Suśruta is to be honored for having built the temple of ophthalmic science in the prehistoric age, von Graefe and others are equally to be honored as the architects of modern ophthalmology, while the illustrious names of Beer, Arlt, Saemisch, Fuchs, Daviel, Knapp, Smith, Barraquer and Wright and others will ever be remembered and glorified as master builders of the edifice of ophthalmic surgery.

DICTYOMA IN AN EARLY STAGE

REPORT OF A CASE

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In 1908 Fuchs [1] gave the name of dictyoma to a malignant tumor of the ciliary epithelium which had the structure of the embryonic retina. As the cardinal properties of this tumor he described:

1. Cellular membranes consisting of cylindric epithelium, with the nuclei arranged in two or more rows, and their axes standing vertically to the surface of the membrane.

2. On the free surface of these cellular membranes a structureless membrane corresponding to the membrana limitans interna of the retina.

3. Cellular membranes consisting of one row of cells, their nuclei arranged in a single row.

4. Solid masses of cells, much like those found in glioma retinae, and rosettes.

5. Infiltrative growth of the tumor, especially into the sclera and the cornea.

Fuchs himself collected 4 cases from the contemporary literature. These were the cases described by Badal and Lagrange [2] (1892), Emanuel [3] (1900), Verhoeff [4] (1904) and Kuthe and Ginsberg [5] (1905). Since then 8 other cases have been described by Greeves [6] (1911), Velhagen [7] and his associates (1917), Satanowsky [8] (1928), Böck [9] (1929), Soudakoff [10] (1936), Shepkalova [11] (1938), Satanowsky and

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11. Shepkalova, V. M.: Vestnik oftal. **13**:95, 1938.

Bernasconi Cramer [12] (1939) and Klien [13] (1939), a total of 12 cases. It should be mentioned here that Greeves himself did not consider the tumor in his case a dictyoma, but both Ginsberg [14] and Gilbert [15] stated that it was of this type.

Because of the small number of cases described and because the dictyoma to be described was seen at a relatively early stage, the following case should not be without interest.

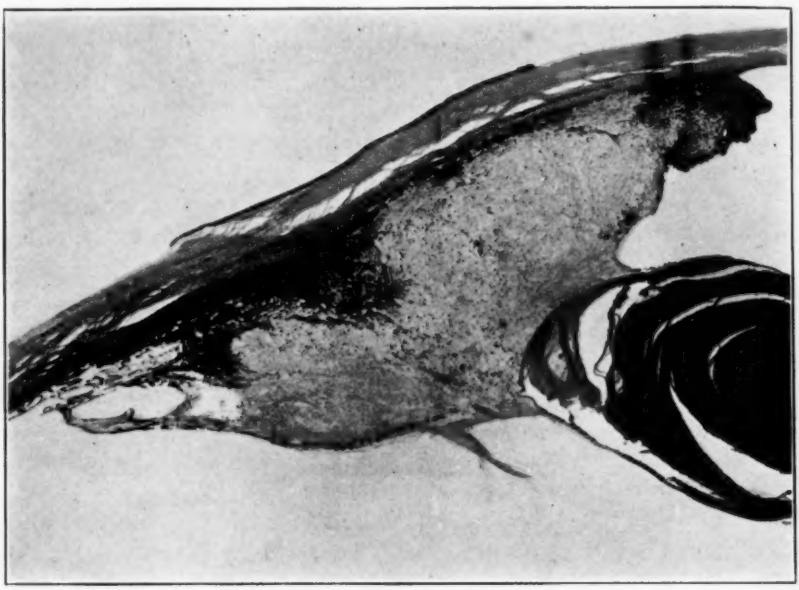

Fig. 1.—Section through the tumor.

REPORT OF CASE

The eye of a 2 year old boy was sent to this clinic for histologic examination after removal for intraocular tumor, cataract and secondary increase of tension. No further details were available.

Histologic examination revealed that the eyeball was of about the normal size. The anteroposterior diameter was 22 mm.

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12. Satanowsky, P., and Bernasconi Cramer, E.: Arch. de oftal. de Buenos Aires **13**:523, 1939; abstracted, Zentralbl. f. Ophth. **43**:69, 1939.

13. Klien, B. A.: Diktyoma Retinae, Arch. Ophth. **22**:432 (Sept.) 1939.

14. Ginsberg, S.: Uvea, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 1, p. 555.

15. Gilbert: Erkrankungen des Uvealtraktus, in Axenfeld, T., and Elschnig, A.: Handbuch der gesamten Augenheilkunde, Berlin, Julius Springer, 1929, vol. 5, pt. 2, chap. 6.

In the deeper layers of the cornea, among the lamellae, rows of cells were seen, mostly in the parts near the limbus.

The anterior chamber was shallow. There were anterior peripheral synechiae. The iris was thinner than usual and of somewhat more compact structure. There

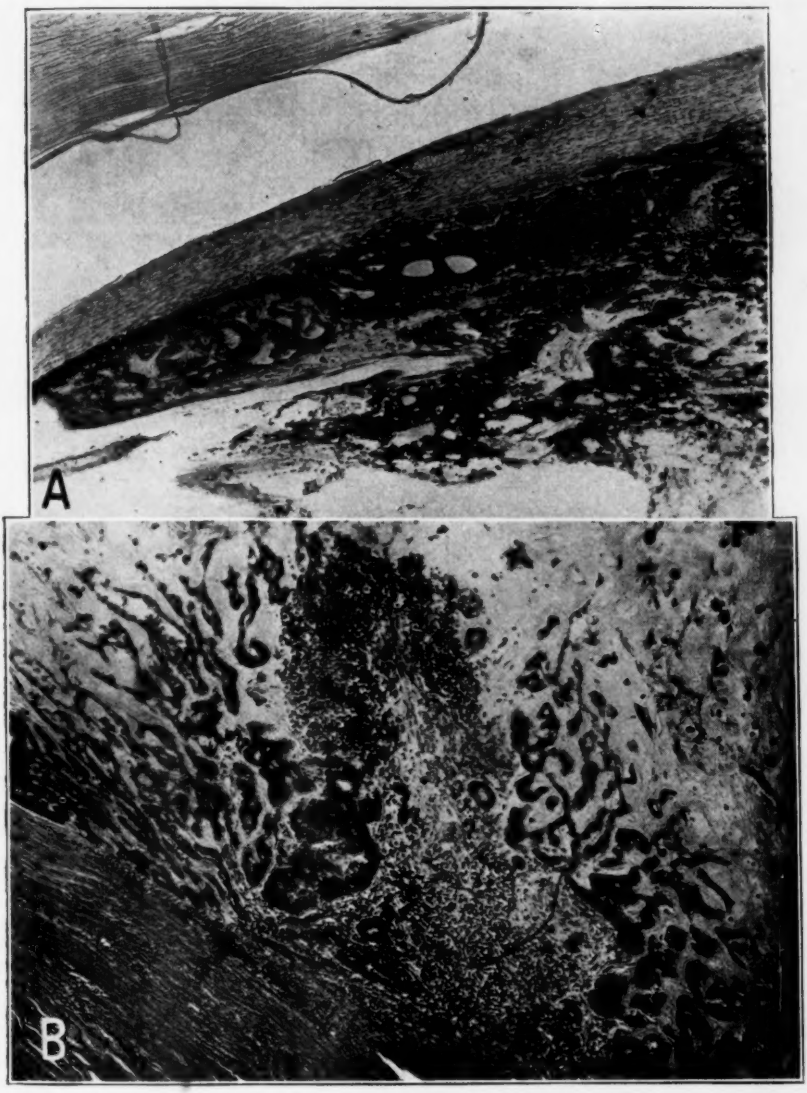

Fig. 2.—*A*, the two structural elements of which the tumor is composed, solid cell masses and cellular membranes. *B*, cellular membranes. In the middle there is a solid mass of tumor cells.

was eversion of the pigment epithelium. There was no hyperemia. In places the pigment epithelium was loosened.

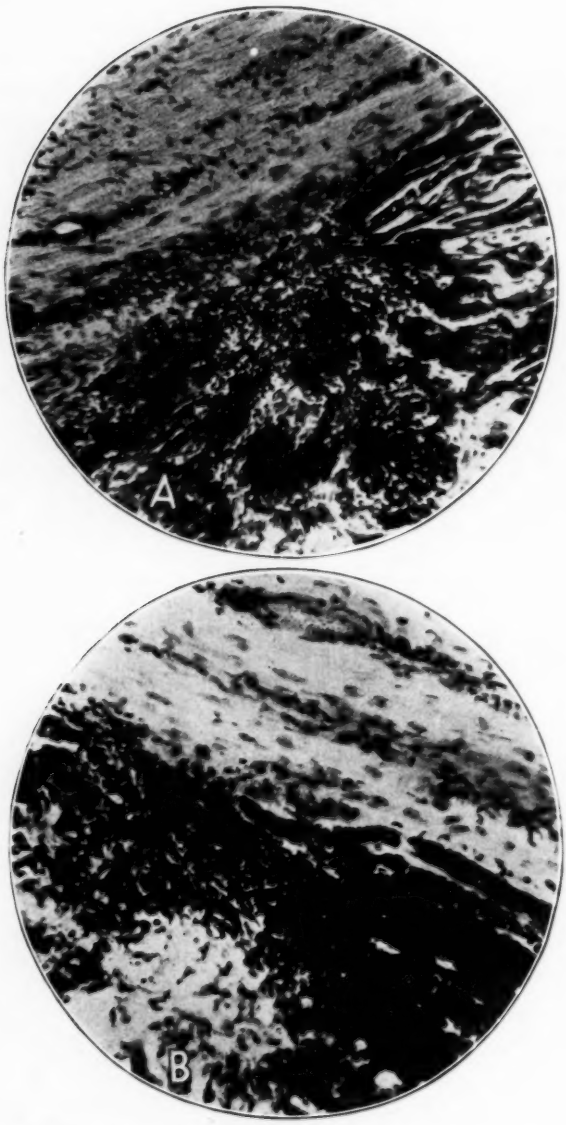

Fig. 3.—*A*, infiltration of tumor cells into the sclera. *B*, cells of the solid cell mass break through a cellular membrane to pass into the sclera. The cellular membrane runs along the sclera but does not enter it.

The ciliary body was elongated. The muscle fibers which ran parallel with the sclera were in the majority. There were only a few ciliary processes.

In other parts, however, the iris and the ciliary body could not be identified. Their place was taken by a tumor advancing from the direction of the ciliary body and taking up the space which is occupied under normal circumstances by

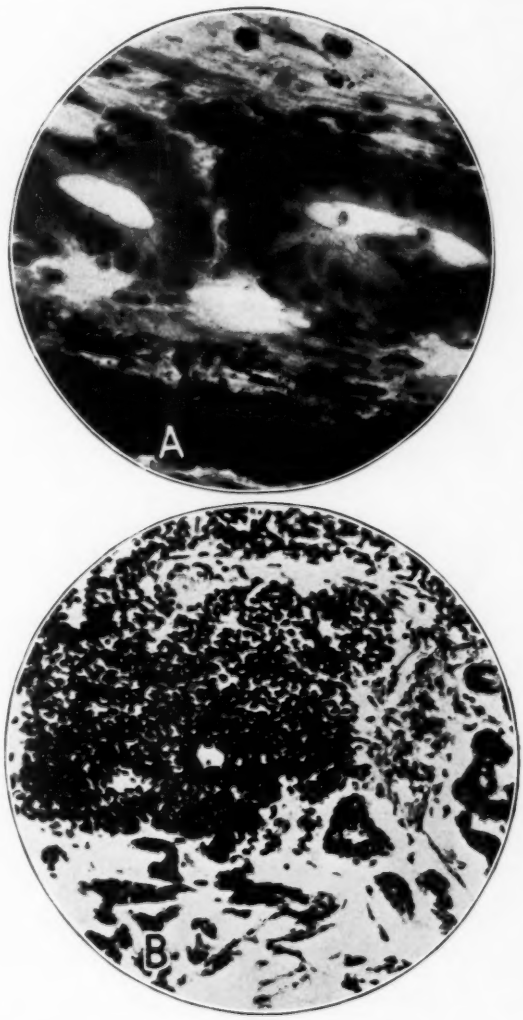

Fig. 4.—*A*, membrane-like structural element on the free surface of the cellular membranes. *B*, solid mass of tumor cells, showing two rosettes.

the iris, the ciliary body and the posterior chamber. The whole tumor was roughly about the size of a normal adult lens. Of this tumor the parts near to the cornea and sclera were constituted chiefly of cells, while in the parts lying nearer to the lens and the vitreous the cells were scarcer. The parts rich in cells showed a

varying structure, but they were constituted of two entirely different types of cell formations: cellular bands consisting of cylindric epithelium and solid groups of cells. In most cases the cellular bands were made up of one row of cylindric epithelium with the nuclei arranged in one or more rows. These cellular bands were actually cellular membranes consisting of one layer of cells, but in cross section they appeared as bands. These cellular bands gave certain parts of the tumor an adenoma-like appearance as they wound to and fro. At some places the cylindric epithelium went over into cuboidal epithelium. On the free surface of these cellular bands a very fine membrane-like structure could be identified. Whether it actually was a separate membrane or simply a condensation of the protoplasm would be very hard to tell. Beside these cellular bands were solid masses of cells, much like those found in glioma retinae. These masses were of various sizes and forms. Often the cells forming them were arranged so as to form rosettes. These cell masses were embedded in connective tissue, in which

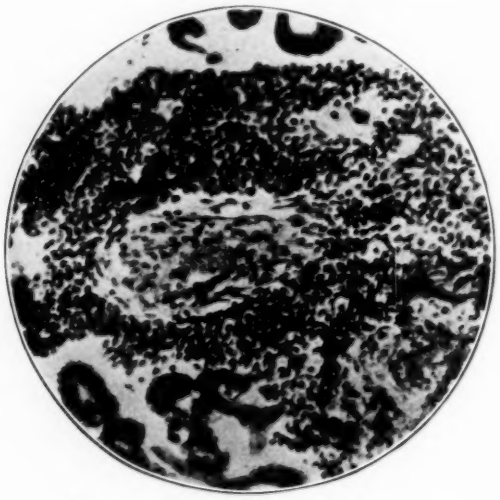

Fig. 5.—Arrangement of glioma-like solid cell masses around a blood vessel.

cells containing pigment were irregularly strewn about. At certain places these pigmented cells were arranged so that they showed where the pigment epithelium of the ciliary body and the iris originally had been. Under the tumor, at the beginning of the pars plana, the pigment epithelium was easily discernible, but on going farther forward it was seen that the tumor broke through the pigment epithelium at many points.

The original structure of the iris in these parts, i. e., the parts occupied by the tumor, was recognizable only at the pupillary margin for about 0.5 to 1 mm. In this small part one could distinguish the sphincter, the stroma of the iris and the pigment epithelium. Otherwise the structure was not recognizable, as it was penetrated by the tumor.

At the corneoscleral junction the tumor went over into the sclera without any special line of demarcation.

Those parts of the tumor which bordered on the lens and the vitreous were much poorer in cells, but at the extreme edge there were again more cells. The cells of this part had more of the character of connective tissue and had processes.

At the extreme edge, however, epithelial tumor cells were again present. Blood vessels were relatively scarce throughout the tumor. Around some of the blood vessels glioma-like solid cell masses were grouped.

In the part of the tumor bordering on the vitreous there was a necrotic area not far from the surface. There was no staining of the nuclei, but cellular debris was easily distinguished.

The tumor had also dislocated the lens and covered part of both the front and the rear surface of the lens—much more, however, of the former than of the latter. In this part of the lens the original structure could not be distinguished. The fibers of the lens were loosened, some of them being broken, and this part stained much more lightly than the rest. Some Morgagni globules were also to be found.

The choroid was normal. There were no special alterations of the retina. The layers of the retina were easily distinguishable. In some places edema of the retina was seen, especially in the layer of cones and rods. The ganglion cells were few and pyknotic. There was excavation of the papilla, and the lamina cribrosa was strongly concave.

COMMENT

To the question whether the tumor was malignant or not I must reply in the affirmative. I consider the following points proof of its malignancy. 1. The tumor cells, especially those of the solid cell groups, were polymorphic. 2. The tumor was rich in cells. 3. Infiltrative growth was observed. 4. The tumor did not respect the boundaries of the different tissues.

It must, however, be mentioned that I could not find any mitotic forms in any part of the tumor. A possible explanation for this might be that the eye was first put into fixing fluid a long time after the enucleation. This is made more probable by the fact that the finer details of the chromatin structure were not discernible.

Summarizing all these observations, the final diagnosis was: dictyoma of the ciliary body, cataract and secondary glaucoma.

Thus only the histologic examination showed that the intraocular tumor in question belonged to the type of malignant tumor of the ciliary epithelium which Fuchs named dictyoma because of the resemblance of the structure of the cellular bands to that of the embryonic retina. Besides the characteristics mentioned at the beginning of this treatise, Fuchs also said that the tumor was to be found only in patients in the first years of life. However, in 1929 Böck described a case in which a dictyoma was found in a 16 year old girl, and in 1936 Soudakoff reported occurrence of the tumor in a 28 year old man. In 2 cases out of 10 the age was well above the first decade, but in the rest the patient was under 10 years of age; so, even though it cannot be said that dictyoma is found exclusively in children in their first decade, it can still be said that it is found in the great majority of cases in children under 10.

The age of 2 years in my case is, as far as I can determine, the earliest age at which dictyoma has yet been found. Otherwise my case did not differ in any important respect from the other cases reported.

However, there is one interesting observation which I should like to mention here, as I have not found it mentioned anywhere else. When examining the slides to determine the malignancy of the tumor, I had the definite impression that the cells of the solid cell masses were more malignant than those of the cellular bands. This difference was most pronounced where the compact tissues of the sclera and the cornea were concerned. Here the cellular bands grew along the boundaries of the sclera and the cornea, so that at some places the cells of the solid cell groups had to break through the cellular bands to be able to infiltrate the sclera and the cornea.

This difference, however, is to be expected when one takes into consideration that the cells which constitute the solid cell groups are much less differentiated and more polymorphic than those of the cellular bands. Thus one may conclude that the former type of cells is more malignant than the latter, because the former is the more immature.

The relation of dictyoma to glioma retinae is not yet clear. But in 1 interesting case mentioned by Fuchs there was a glioma growing from the pars optica retinae, and a tissue which resembled the embryonic retina in structure was found on the pars caeca. It is probable that thorough examinations of such atypical conditions will bring the solution to many a question concerning dictyoma.

ORBITAL IMPLANTS

WITH SPECIAL REFERENCE TO VITALLIUM

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NEW YORK

The tolerance of the orbital tissue to foreign materials has always been well known, and for this reason many substances have been implanted in Tenon's capsule. Most ophthalmologists of the present day concede that something should be put into the socket after enucleation in order to obliterate the unnatural folds and depressions in the upper and lower lids and to prevent ptosis, impaired motility and sinking of the artificial eye.

For this reason aluminum, celluloid, sponge, wool, silver, rubber, silk, catgut, peat, agar agar, paraffin, asbestos, cork, ivory and other substances have been used, with varying success. At present the most common implants are gold balls, glass balls, fat and fascia lata. Each of these has its champions, and I am sure there is no ophthalmologist who has not experienced the unpleasant sensation of finding an implant expelled from the orbit for no apparent reason. The operation seemed perfect; there were no complications; nevertheless, the implantation was a failure.

During the World War, Magitot, after the successes of numerous general surgeons with formaldehyde-treated bone cartilage, adopted it for orbital implantations, and after my return I used it with considerable success at Bellevue Hospital. The material was hard to obtain; so I applied to a large, well known pharmaceutic house for assistance. The staff seemed interested but after referring the matter to their experimental department reported that there was some danger of anthrax infection. There is now on the market, however, an implant made of cartilage which seems to have a great number of champions. In 1 case in which I operated the formaldehyde-treated cartilage implant seemed to be infected, or chemical changes had taken place in the socket, five months after enucleation. I thought best to remove it. This was rather difficult, as Tenon's capsule was thickened and seemed actually to send proliferations into the cartilage. The failure, I believe, was due to improper fixation and may have resulted from the action of the formaldehyde.

Formaldehyde-treated beef cartilage does after some time absorb (fig. 1). The late Dr. J. M. Wheeler, in an article in the ARCHIVES OF

OPHTHALMOLOGY, mentioned making implants of cork and stated that "a peculiar phenomenom made their use inadvisable. Long after their implantation the tissues in front of them sloughed, and they appeared in the cul-de-sac. Why this happened I do not know, but I suppose that some chemical change took place with a degree of disintegration of the cork, and necrosis of the tissue about the cork." Glass has not proved entirely satisfactory. Dr. Clyde E. McDannald reported a glass implant which shattered in the orbit, apparently as the result of erosion. I know of a similar case. All ophthalmologists have seen artificial eyes in which erosion has taken place. Fat has not proved satisfactory in all cases. There have been liquidation of the fat, shrinkage and occasionally the formation of a lipoma. While gold seems to be most suitable, the gold

Fig. 1.—*A*, roentgenogram taken eighteen years after implantation of a formaldehyde-treated beef cartilage implant. The case was originally reported in the *American Journal of Ophthalmology* for January 1923. *B*, roentgenogram (same case) showing the formaldehyde-treated beef cartilage. Note the absorption of the cartilage.

alloys sometimes contain only 15 per cent of pure gold and are therefore liable to chemical decomposition. The question as to which substance that has been tried is the best material for orbital implantation resolves itself into another question: Is there a material which will remain chemically (and possibly electrically) neutral in body fluids and will remain entirely silent in the body, apparently creating no change in tissue or in bone?

As metallurgic knowledge increases, particular alloys are developed for specific fields and in their applications exhibit advantages of superior properties not obtainable in the metals of which they are composed. For

this reason special alloys are being produced to meet the ever increasing needs in industry for finer, better and more efficient materials. I wish to call attention to an alloy called vitallium, a product of the Austenal Laboratories, Inc.; to the medical uses to which it has been applied, and to the clinical and scientific experiments that have been performed with it. Vitallium has been used in prosthetic dentistry for a long period. That saliva is an electrolyte there can be no question, and galvanic action occurs in the human being from time to time, especially when dissimilar metals are used in the oral cavity for filling, crowns and restorations. When vitallium is used in combination with other metals this galvanic action is much reduced. With this in mind, Dr. Charles S. Venable,

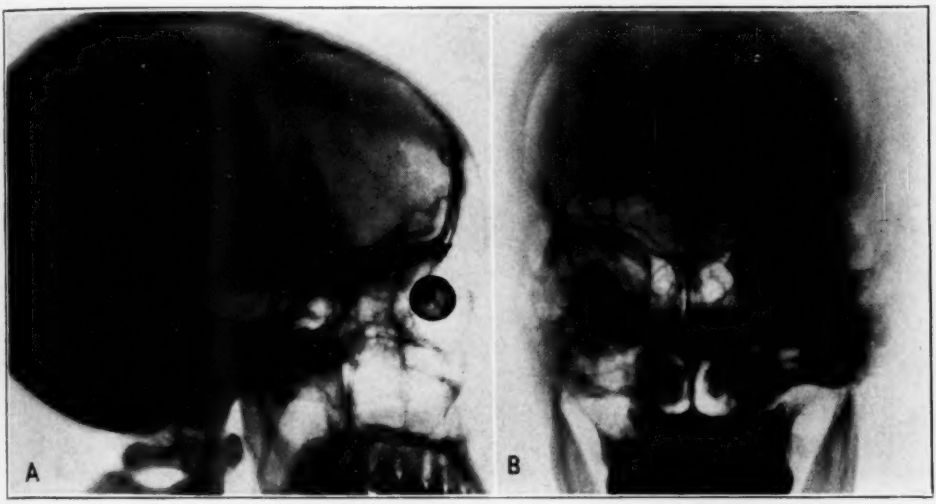

Fig. 2.—*A*, roentgenogram showing a vitallium implant in Tenon's capsule and perfect apposition of the artificial eye. *B*, front view of a vitallium implant in Tenon's capsule. Note the perfect position of the implant in the orbit.

of San Antonio, Texas, conducted a series of experiments on dogs to establish the reactions between the body fluids and metals. He concluded that in dealing with fractures an alloy should be sought and developed that is entirely inert in the presence of, and unaffected by, biologic salts to the extent that there will be practically no electrolytic action and that it should have the strength to resist such stress or strain as it may have to bear. He accordingly anesthetized dogs, placing two screws consisting of the various metals and alloys in the bone about 1.5 cm. apart. In all the animals the right radius was fractured and the left radius left intact in order to check the reaction in each dog with and without trauma of the bone. Before the screws were placed, drill holes about two-thirds the size of the screws were made in order to obviate

the possibility of undue or variable pressure. After a large number of prolonged and exacting experiments with different metals he reached many conclusions, but he stated that with vitallium there was no tissue reaction or osseous change at the site of any screw, though many experiments were performed with different combinations of metals. Vitallium is a casting alloy, 90 per cent of which is composed of cobalt and chromium, with a smaller percentage of molybdenum. It is designated as a cobalt-chromium alloy. It is exceptionally strong, hard and very light. It has a specific gravity of 8.29, which is much less than the specific gravity of pure gold, which is 19.3, or of casting golds, which ranges from 14.0 to 18.0. I wish to emphasize the difference in weight between a vitallium implant and a gold implant. Vitallium resists strong mineral acids, and a solution of sodium chloride of any degree of concentration over any period produces no effect on it. Vitallium is stronger than gold; it is cheaper than gold; it *can be boiled,* and it lies *inert* in the body tissues. Any implant in the orbit certainly should be sterilized by boiling. Vitallium produces no tissue reaction, and it is not influenced by heat or by cold as are other synthetic and plastic materials. I have used vitallium orbital implants for over a year and in 6 cases have had no complications and excellent cosmetic results. Some of my colleagues have had similar successes.

Many operators have reported unusual successes in the treatment of fractures with surgical nails, screws, plates, hip caps and special appliances made of vitallium. This material has been used to make a prosthesis when a squamous cell carcinoma on the dorsum of the nose made surgical removal of the nose necessary because surgical restoration was not considered advisable. It has been used to replace parts of the human skull and face, and sufficient time has elapsed to warrant that it has justified the prognosis associated with its use. The disfigurement caused by facial deformities secondary to the removal of malignant tumors of the face has been greatly lessened.

The shapes of orbital implants (fig. 3 *A*) certainly are worthy of attention. While experimenting with beef cartilage I tried out a cartilage implant that was practically spherical; that is, the anterior third was cut off flat, the idea being to obtain better motility. In a number of cases the implants became misplaced in Tenon's capsule, no doubt because of the action of the ocular muscles, with resulting strabismus on fitting of the artificial eye. I do not believe that this failure was due to using too small an implant. It is my belief that cone-shaped implants by the presence of their tails in Tenon's capsule limit motion in any direction. The eyeball is normally nearly a sphere, and if it is properly enucleated a spherical implant is the best. In my experience other shapes either become displaced or produce limitation of motion of the

artificial eye. The external surface of the implant should by all means be dimpled (fig. 3*B*), as this materially assists in securing it firmly in Tenon's capsule.

An important detail in any enucleation is the use of the strabismus hook, which should be handled with extreme care. There is much danger of puncturing Tenon's capsule and producing a rent or a tear, and in the presence of a tear, owing to the action of the ocular muscles, the implant slips through the opening and is eccentrically placed in the

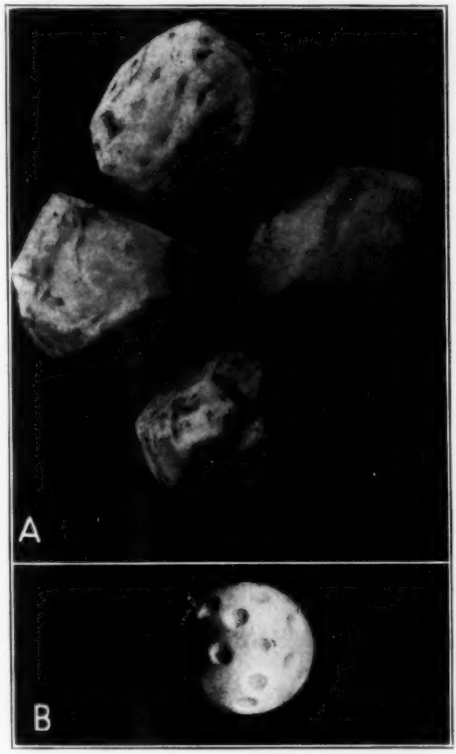

Fig. 3.—*A*, various models of formaldehyde-treated beef cartilage implants used in Bellevue Hospital in 1922. *B*, vitallium orbital implant. The dimpled surface helps to secure the implant in Tenon's capsule and will cause no irritation as might a surface with elevated points.

orbit, with annoying deformity. Figure 2*A* illustrates the correct position of the orbital implant in Tenon's capsule and its relation to the orbital margin. Figure 2*B* depicts the proper apposition of the artificial eye and the implant.

The dissection should be done entirely with the scissors. Afterward it is permissible to pass the strabismus hook around the globe backward to ascertain whether there are any adhesions that have not been dissected.

Another important fact to remember is that a pressure dressing should be kept on for at least ten days to two weeks to obtain consistent results and proper healing.

From a summary of the reports of experimental and clinical uses that have been made of vitallium, backed by recognized authorities, this substance seems to me to be the one most suited for orbital implantation, and it is my opinion that a globe-shaped implant will give the best cosmetic result, with the least limitation of movement of the artificial eye.

The Austenal Laboratories, Inc., and Dr. Charles S. Venable, of San Antonio, Texas, cooperated with me in this study.

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CAUSES OF BLINDNESS IN HAWAII

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In any state or territory a comprehensive survey concerning the causes of blindness is the first real step toward the preservation of human eyesight. A study of this type to be uniform should be compiled entirely from the reports of recognized ophthalmologists; to be complete it should include data on the blind from all of the economic stratums of society; to be scientifically accurate it should consider etiologic as well as topographic groupings, and lastly, to have other than medical (e. g., social) significance it should take into account the racial, geographic, climatic and industrial conditions peculiar to the various communities.

The statistics which follow were obtained in a recent survey conducted among the blind of the Territory of Hawaii. The classifications adhered to and the special report forms used in compiling the data were recommended by the Committee on Statistics for the Blind. The data were secured from the records of 379 charity and private patients. Of these 340 were examined by ophthalmologists. The remaining 39 were persons known to be blind who had been repeatedly examined by medical social workers trained for ophthalmologic work, but who refused to return for a check-up by an oculist. Each record was carefully scrutinized by the members of the Territorial Review Committee for the Blind. Only data for patients who were approved by all three members of this committee and whose visual state conformed to the definition of economic blindness set up by the Section on Ophthalmology of the American Medical Association were included in the etiologic and topographic tables. The group of persons known to be blind [1] constituted less than 0.1 per cent of the total population of the territory; it included representatives from all of the principal races and racial mixtures found on the eight islands comprising the Hawaiian group.

1. While this paper was being prepared, approximately 20 additional blind or nearly blind patients came to our attention. Data for them have not been included in this study because at this time there is insufficient information from the ophthalmologist or the social worker concerning their ocular status. The number of blind persons residing in Hawaii at present is estimated at 400.

DISTRIBUTION OF THE BLIND IN HAWAII BY AGE AND SEX

The most striking feature of this table was the constantly increasing incidence of blindness with age from infancy to senility.

Blindness was found to a greater extent in the male than in the female population of the territory. This may be ascribable to the

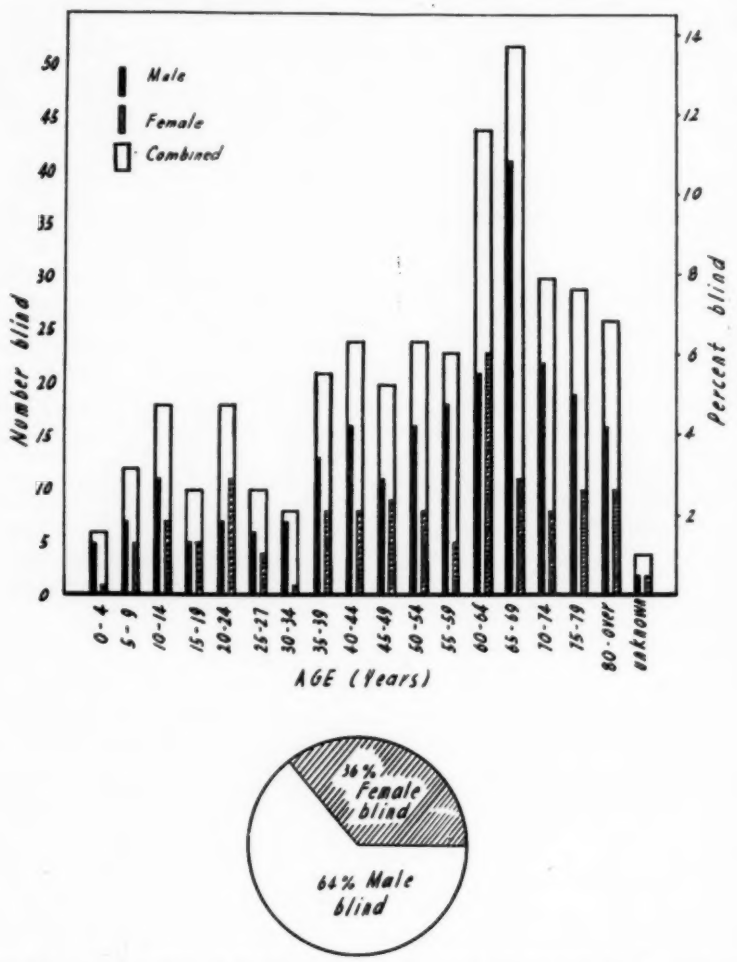

Chart 1.—Distribution of the blind in the Territory of Hawaii by age and sex.

male's greater liability to industrial hazards and exposures, to his greater tendency toward venereal diseases and to the locally existing ratio of 1.5 men to 1 woman.

RACIAL CLASSIFICATION OF THE BLIND IN HAWAII

Special attention was paid to the racial extraction of the patients because it was hoped that valuable information might be secured by

correlating the occurrence of specific diseases of the eye with race. Ancient religious rites, tribal customs and traditions kept alive and adhered to by descendants of various races were found to be responsible for a large number of ocular tragedies which might have been averted by early judicious medical intervention.

ETIOLOGIC CLASSIFICATION OF BLINDNESS IN HAWAII

It was impossible to determine the actual etiologic factor in each instance of blindness. Several patients with shrunken deformed bulbs or with empty sockets were seen years after the condition primarily

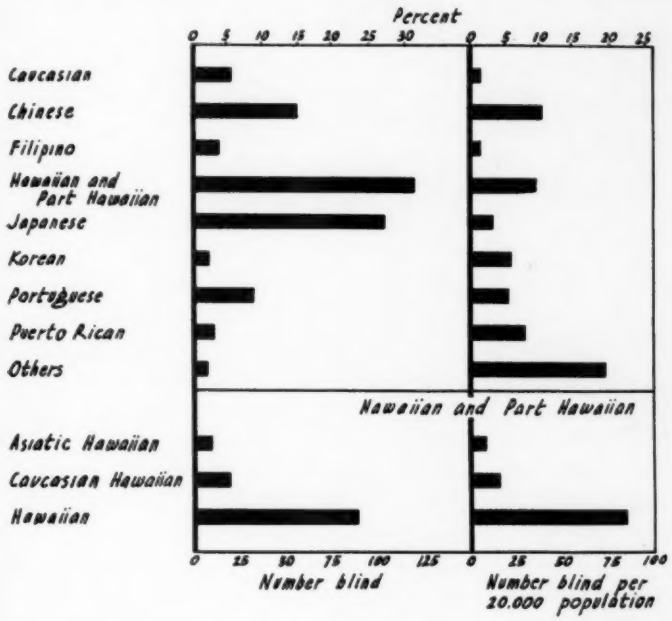

Chart 2.—Distribution of the blind in the Territory of Hawaii by race.

responsible for their blindness had abated. Inquiries by social workers elicited the exciting cause in a number of these instances, but often the diagnosis was a matter of conjecture, arrived at by piecing together fragments of inaccurate statements given by the patient, his relatives or his friends.

In the infectious disease category syphilis headed the list. It took its heaviest toll from the Hawaiian group. The high incidence determined for this disease (12.36 per cent) corresponds to figures obtained elsewhere. Cowan and Sinclair [2] reported that of 6,000 applicants for pensions for the blind in Pennsylvania 9.4 per cent had syphilis. Berens,

2. Cowan, A., and Sinclair, S. M.: Causes of Blindness in Pennsylvania, J. A. M. A. **107**:757 (Sept. 5) 1936.

Kerby and McKay [3] reported an incidence of syphilis of 4.7 per cent in a study of 2,702 blind school children.

Leprosy was the second commonest cause of blindness in the infectious disease group. The ocular ravages of this disease have been

TABLE 1.—*Causes of Blindness in Hawaii: Etiologic Classification*

Cause of Blindness	Racial Distribution of Blindness									Total Number	Percentage
	Caucasian	Chinese	Filipino	Japanese	Korean	Portuguese	Puerto Rican	Hawaiian and Part Hawaiian	Other Races		
Infectious diseases											
Measles	..	..	..	..	..	..	..	1	1	2	0.59
Meningitis	..	..	..	..	..	..	1	1	..	2	0.59
Ophthalmia neonatorum											
Gonorrhea	..	2	..	1	..	1	..	..	..	4	1.17
Other types, specified	..	..	1	..	..	..	..	..	..	1	0.29
Other types, not specified	..	..	..	1	..	..	..	2	..	3	0.88
Syphilis	..	..	..	1	..	..	..	2	..	3	0.88
Prenatal	..	..	..	4	..	..	..	4	..	8	2.35
Acquired after birth	4	3	..	6	1	2	..	12	1	29	8.53
Type not specified	..	..	..	2	..	..	..	..	..	2	0.59
Trachoma	..	11	4	12	..	..	..	1	1	29	8.53
Tuberculosis	..	..	1	..	..	2	..	1	..	4	1.17
Leprosy	1	..	..	5	..	3	..	24	..	33	9.69
Others, specified	1	..	..	1	..	..	..	..	..	2	0.59
Not specified	..	1	2	3	1	2	..	4	..	13	3.82
Trauma											
Nonoccupational activities	..	..	..	1	..	..	..	..	..	1	0.29
Birth processes	..	..	..	1	..	..	..	..	..	1	0.29
Medical and surgical procedures	1	2	..	3	..	..	1	..	..	7	2.06
Play or sport	1	..	..	..	..	..	..	..	..	1	0.29
Occupational activities *	..	1	..	2	2	1	..	6	1	13	3.82
Other activities, specified	1	1	..	..	..	..	1	1	..	4	1.17
Activity not specified	1	..	..	1	..	..	..	1	..	3	0.88
Poisonings											
Nonoccupational activities	..	..	..	1	1	..	..	1	..	3	0.88
Neoplasms											
All types	..	..	..	1	..	2	..	..	..	3	0.88
General diseases											
Diabetes mellitus	..	..	..	1	..	..	..	3	..	4	1.17
Nephritis	..	2	..	..	..	..	1	..	..	3	0.88
Vascular diseases	6	1	..	3	..	1	3	2	1	17	5.00
Diseases of the central nervous system	..	1	..	..	..	..	..	..	..	1	0.29
Nutritional deficiencies	..	..	..	..	..	1	..	..	..	1	0.29
Others, specified	1	..	..	1	..	..	..	..	..	2	0.59
Blindness of prenatal origin											
Hereditary origin established	2	3	2	15	..	6	..	6	..	34	10.00
Hereditary origin presumed	..	..	1	5	..	1	..	..	..	7	2.06
Cause undetermined											
Unknown to science	1	15	2	8	2	2	3	20	2	55	16.16
Undetermined by physician	1	5	2	9	..	2	2	15	1	37	10.88
Not specified	2	2	..	1	..	1	..	..	..	6	2.35

* Dynamite explosions, such as in fishing, mining, road building and welding.

previously emphasized by other investigators who called attention to the frequency of blindness among patients with leprosy. Recently Prendergast,[4] in a report on the ocular status of 350 inmates of the National Leprosarium in Carville, La., stated that 20 of them were

3. Berens, C.; Kerby, C. E., and McKay, E. C.: The Causes of Blindness in Children, J. A. M. A. **105**:1949 (Dec. 14) 1935.

4. Prendergast, J. J.: Ocular Leprosy in the United States, Arch. Ophth. **23**:112 (Jan.) 1940.

blind in both eyes. This figure approximates ours; we found that 33 of the 443 lepers in Hawaii were blind. The disease was originally brought to the islands by Chinese immigrants and was referred to as the "Chinese disease." At present the largest number of blind lepers are of Hawaiian and part Hawaiian ancestry.

Trachoma, which ranked third highest among the communicable diseases causing loss of sight, was responsible for 8.53 per cent of the blindness in the population. It also had filtered into Hawaii from the Orient and was distributed equally between the Chinese and the Japanese settlers. The ravages and destructive influences of trachoma are well known throughout the world. MacCallan [5] recently presented comprehensive statistics concerning its universal distribution. In Kentucky 15 per cent of 8,444 patients with trachoma were blind. In Missouri 12 per cent of the population with trachoma were found to have visual acuity of less than ability to count fingers at 2 meters. Gradle and De Francois [6] reported that of a group of 2,713 patients with trachoma in Illinois 7.8 per cent were industrially blind.

Gonorrheal ophthalmia accounted for 2.34 per cent of the blindness in our group of cases. This figure is extremely low and compares favorably with the much higher incidence of blindness from this cause in other localities. The National Society for the Prevention of Blindness [7] reported a yearly constant for the past eight years of 7 per cent for blindness caused by ophthalmia neonatorum among new students seeking admission to schools for the blind throughout the country. It is more than likely that many patients who were placed in the congenitally blind group by us in reality had gonorrheal ophthalmia at birth.

Traumatism of all sorts was responsible for blindness in 30 cases. In 13 cases blindness was caused by dynamite explosions which occurred during road building and fishing. Ocular injuries caused by cane leaf and pineapple needles did not figure prominently in the causation of permanent visual disability. The rigid enforcement of protective appliances for those engaged in hazardous occupations was responsible for this enviable record.

A diagnosis of neoplasm was made in only 0.88 per cent of the cases in our group. This figure is lower than similar ones reported elsewhere. Ubaldo and Ocampo,[8] on the basis of data from the files of the Philippine

5. MacCallan, A. F.: The World-Wide Distribution of Trachoma, Brit. J. Ophth. **22**:513 (Sept.) 1938.

6. Gradle, H. S., and De Francois, W.: The Visual Ravages of Trachoma, J. A. M. A. **109**:253 (July 24) 1937.

7. Prevention of Blindness from Ophthalmia Neonatorum, 1939 Report of Standing Committee on Conservation of Vision, State and Provincial Health Authorities of North America, Sight-Saving Rev. **9**:126 (June) 1939.

8. Ubaldo, A. R., and Ocampo, G. E.: Blindness in Children, J. Philippine Islands M. A. **19**:483 (Aug.) 1939.

General Hospital, reported that intraocular tumors figured fourth among the causes of loss of sight in children under 14 years of age.

Vascular diseases were noted in 17 cases. The incidence of this group of derangements was highest in the Occidental population. The generally more stable makeup of the Oriental peoples has been advanced as an explanation for their relative freedom from the cardiovascular-renal diseases and the blindness caused by them.

The incidence of blindness of prenatal origin was unduly high. It is more likely that if thoroughly accurate medical histories could have been obtained many cases of blindness presumed to be due to developmental anomalies would have been reclassified under other specific headings.

TOPOGRAPHIC CLASSIFICATION OF BLINDNESS IN HAWAII

Glaucoma of the adult type was found only in 6.86 per cent of all cases. It occurred less commonly in the Oriental than in the Occidental races. The close relation between emotional upsets and the outbreak of acute glaucomatous crises might in part explain the infrequency with which primary glaucoma was encountered among the more placid and phlegmatic Oriental peoples.

Myopia which resulted in loss of sight occurred infrequently (in 1.76 per cent of the cases), but it was a common refractive error. It was seldom of the progressive and malignant variety.

Albinism was extremely rare (present in 0.29 per cent of the cases). This was unexpected because the syndrome is generally more common among the dark-skinned races.

Pterygium was listed as the cause of blindness in 2 (0.59 per cent) of the cases. In both cases the patient was an elderly, uncooperative Oriental man who lived in an outlying district where facilities for transportation and for medical care were poor.

Diseases of the cornea were the most frequently diagnosed ocular derangements which resulted in blindness. Syphilis, leprosy and trachoma, all diseases with a predilection for involvement of corneal tissue, were responsible for the high incidence of cases in this category.

Iritis and iridocyclitis occurred in 2.94 per cent of the cases. This percentage is compatible with the small number of cases of endogenous iritis met with in routine ophthalmologic practice in the Territory of Hawaii.

Cataracts were noted in 20 per cent of the blind population. No effort was made to determine the role played by infection, intoxication, trauma or senescence in the production of cataracts. Elliott [9] ascribed the prevalence of cataracts in the tropics to the effect of excessive light

9. Elliott, R. H.: Tropical Ophthalmology, London, Oxford University Press, 1920, p. 285.

on the lens and to the special diet of the people. Meesmann [10] expressed the belief that sclerosis of the nucleus and senile opacities in the cortex are merely symptoms of biologic aging of the patient.

TABLE 2.—*Causes of Blindness: Topographic Classification*

Cause of Blindness	Racial Distribution of Blindness									Total Number	Percentage
	Caucasian	Chinese	Filipino	Japanese	Korean	Portuguese	Puerto Rican	Hawaiian and Part Hawaiian	Other Groups		
Eyeball											
General causes											
Hypertension	4	4	1	6	..	5	3	3	..	26	7.64
Myopia	2	2	1	1	..	..	..	..	..	6	1.76
Panophthalmitis	..	1	..	1	..	..	..	1	..	3	0.88
Anomalies											
Albinism	..	..	..	1	..	..	..	..	..	1	0.29
Megalophthalmos	..	..	..	..	..	2	..	..	..	2	0.59
Microphthalmos	..	..	..	2	..	..	..	..	..	2	0.59
Coloboma	..	..	1	..	..	..	..	..	..	1	0.29
Others, specified	1	..	..	..	..	1	..	..	..	2	0.59
Degenerative changes											
Disorganized eyeball	..	3	1	4	1	1	..	3	2	15	4.41
Other conditions, specified	..	..	1	..	.	1	..	..	..	2	0.59
Not specified	..	..	1	..	..	..	..	..	..	1	0.29
Conjunctiva, muscles											
Pterygium	..	1	..	..	1	..	..	..	..	2	0.59
Cornea											
Interstitial keratitis	1	..	..	5	..	..	..	2	..	8	2.35
Phlyctenular keratitis	..	..	1	..	..	2	..	..	..	3	0.88
Ulcerative keratitis	..	1	..	3	..	..	..	2	1	7	2.06
Keratitis of unspecified type	..	2	1	4	..	..	..	4	..	11	3.23
Pannus	..	8	..	3	..	..	..	1	1	13	3.82
Vascular ulceration	..	5	1	4	..	..	..	..	..	10	2.94
Leprous keratitis	1	..	..	5	..	3	..	24	..	33	9.69
Other conditions, specified	1	1	1	4	1	1	..	2	..	11	3.23
Condition not specified	..	..	..	2	..	..	..	1	..	3	0.88
Iris and ciliary body											
Iritis	1	1	2	1	..	..	..	..	..	5	1.47
Iridocyclitis, uveitis	1	..	..	2	..	1	..	1	..	5	1.47
Condition not specified	..	..	..	..	..	..	..	1	..	1	0.29
Crystalline lens											
Cataract	5	12	1	14	1	2	4	26	3	68	20.00
Dislocated lens	..	..	..	3	..	..	1	2	..	6	1.76
Condition not specified	..	1	..	..	..	..	..	..	..	1	0.29
Choroid and retina											
Choroiditis	..	1	..	2	..	..	..	..	..	3	0.88
Retinitis	..	..	..	..	..	1	..	1	..	2	0.59
Chorioretinitis	2	2	1	2	1	3	..	..	..	11	3.23
Detached retina	2	..	..	2	..	..	1	..	..	5	1.47
Retinal degeneration	..	..	1	2	..	1	..	1	..	5	1.47
Other conditions, specified	..	..	..	4	..	..	1	1	..	6	1.76
Optic nerve, visual pathway, cortical visual centers											
Optic nerve atrophy	2	8	1	12	2	4	1	20	1	51	14.99
Optic neuritis	..	..	..	1	..	..	..	..	..	1	0.29
Papilledema	..	..	..	..	..	1	..	..	..	1	0.29
Retrobulbar, intracranial lesions	..	..	1	1	..	..	..	1	..	3	0.88
Other conditions, specified	..	1	..	..	..	..	..	1	..	2	0.59
Site not well defined											
Amblyopia, undefined	..	..	..	..	..	..	..	1	..	1	0.29
Lesions, not specified	..	..	..	1	..	..	..	..	..	1	0.29

Choroiditis and chorioretinitis were responsible for blindness in 4.41 per cent of the cases. This figure does not sufficiently emphasize

10. Meesmann, A.: Klinische Beobachtungen bei Glasmacherstaren und ihre Beziehungen zu anderartigen Starformen, Klin. Monatsbl. f. Augenh. **92**:289 (March) 1934.

the prevalence of acute choroiditis and its local importance in producing unilateral blindness. The disease occurred principally in young adults, who were often in good physical condition. Duggan [11] advanced the theory that a localized weakness of the choroidal vessel walls or a selective vascular sensitivity to some circulating toxin might account for the recurring attacks and the dire sequelae of this disease.

Primary "idiopathic" detachment of the retina was the cause of impaired functional ocular integrity in 1.47 per cent of the cases. In this category were also placed cases of retinal detachment following nonperforating injuries. According to several authors the proportion of patients with bilateral detachment among all patients with detachment is as follows: Gonin,[12] 11.6 per cent; Arruga and co-workers,[13] 17.1 per cent; Dunnington and Macnie,[14] 17.8 per cent; Maertens,[15] 20 per cent, and Gifford,[16] 22.6 per cent. The variation in incidence may be explained by differences in the diagnosis of the condition of the eye first involved made by different authors. The detachment in the eye first affected may often have been hidden by a complicating cataract, or the eye may even have been removed because of painful secondary iridocyclitis.

Invasion of the optic nerves and visual pathways which resulted in atrophy of the papillae occurred in 13.43 per cent of the cases in our series. Under this heading were grouped all cases of atrophy of the optic nerves, irrespective of cause.

COMMENT

This study is submitted with the hope that it will stimulate further medical research on the causes of diseases resulting in blindness, that it will stress the importance of thorough examinations and carefully prepared case reports and that it will further the crusade for sight saving, which is directed toward the reduction and the ultimate eradication of preventable blindness.

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11. Duggan, W. F.: Acute Exudative Choroiditis, Arch. Ophth. **23**:930 (May) 1940.

12. Gonin, in Lagrange, F., and Valude, E.: Encyclopédie française d'ophthalmologie, Paris, O. Doin, 1903-1919, vol. 6, p. 977.

13. Arruga, H.; Ovio, G., and Vogt, A.: Temas officiales (rapports) sobre el desprendimiento de la retina, in XIV Concilium Ophthalmologicum 1933, Hispania, Madrid, 1933, vols. 1 and 2.

14. Dunnington, J. H., and Macnie, J. P.: Detachment of the Retina: Operative Results in One Hundred and Fifty Cases, Arch. Ophth. **13**:191 (Feb.) 1935; Detachment of the Retina: Operative Results in One Hundred and Sixty-Four Cases, ibid. **18**:532 (Oct.) 1937.

15. Maertens, H.: Das andere Auge bei spontaner Ablatio, Inaug. Dissert., Cologne, T. Borowsky, 1937.

16. Gifford, S. R.: Surgical Treatment of Retinal Detachment, Arch. Ophth. **16**:405 (Sept.) 1936.

PRISM COMPENSATION IN CASES OF ANISOMETROPIA

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A moderate amount of anisometropia is frequent and usually does not require any compensation in the lens prescription to insure the patient's comfort. Larger degrees of anisometropia are not uncommon, and when fusion is not suspended compensating prisms are often required to obviate the hyperphoria developed when the visual axes are moved from the primary to the reading or working position. The problem, although frequently overlooked, is familiar to any one doing refraction. It has been well covered from the optical standpoint by Obrig.[1] However, the following facts will bear repetition.

If a 1 D. lens is decentered 1 cm., there is an image displacement of 1 prism diopter. The same phenomenon occurs in spectacle lenses when the visual axes are moved from the primary to the reading position, the power of the prism produced depending on the angular displacement of the visual axes and the power and type of the correcting lens, cylinders at 90 degrees producing no change. The prism developed is base up if the lens is of plus power and base down if the lens is minus in power. If the two lenses are of equal power and sign no hyperphoria is developed. However, when the lens combinations differ for the two eyes a vertical displacement of the images occurs. When the correcting lenses are of low power the patient is usually able to compensate for the small amount of hyperphoria developed and does not have ocular discomfort. Also, a patient frequently can compensate for larger amounts of hyperphoria. However, many patients who require a strong lens combination before one or both eyes need prism compensation to relieve them of early ocular discomfort when reading or doing close work of any type.

Such patients, as a rule, have varied asthenopic symptoms, frequently absent in the morning but increasing as the day progresses. They

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From the Section on Ophthalmology, the Mayo Clinic.

1. Obrig, T. E.: Modern Ophthalmic Lenses and Optical Glass, Philadelphia, Chilton Press, 1935.

usually have had repeated refractions and glasses and one or more courses of ocular exercises without any material improvement in their symptoms. At times they present the picture of an ocular neurosis and complain of constant ocular discomfort in near work.

The question of whether or not an individual patient with a high degree of anisometropia or actual antimetropia should have prism compensation for his near work is one which frequently confronts the oculist. If it is decided that there should be compensation, the usual practice has been to ask the optician to supply this in the form of a compensating prism bifocal or a slab-off. He does this usually on the basis of 8 or 10 mm. of depression of the eyes in near vision.

However, if one uses a Maddox rod at the patient's individual reading position with the correction in place, the findings frequently do not coincide with those which the arbitrary compensation would give in a particular case. This apparently is due to individual variation in the position of near work or reading matter as well as to a tilting forward of the head which is common in this group. It is surprising but not uncommon to find a patient who is comfortable wearing glasses with 5 D. difference in the correction of the two eyes and who has good fusion and stereopsis and no hyperphoria demonstrable with the Maddox rod in downward gaze. These variations have caused us to change our method of handling these patients and to prescribe compensation according to our findings in the individual case.

The following procedure has been adopted when a patient of this type is encountered. The phorias are measured; a refraction is then done with the eyes under homatropine cycloplegia, and the glasses are prescribed to correct the error found at a postcycloplegic examination. Then with the patient wearing his glasses the hyperphoria developed at his individual reading or working position is measured a number of times on succeeding days. A small fixation light is held in the position where the patient ordinarily reads or works. A Maddox rod is held over one eye, and the degree of hyperphoria is measured by holding graduated prisms in front of either eye until the horizontal streak of the Maddox rod passes through the center of the light. Clip-on lenses with divided prisms are then prescribed for the amount which appears indicated by the repeated examinations.

The patient is instructed to wear the prisms for close work. At the end of a month's trial, if the asthenopia has abated, a compensated prism bifocal or slab-off lens is given, which dispenses with the cumbersome clip-ons. If the patient is still not comfortable the amount of prism may be varied and use of the clip-ons continued for another period.

The following cases are illustrative of this condition.

REPORT OF CASES

CASE 1.—A white man aged 37 complained of increasing ocular fatigue when working. He was employed as a secretary in an insurance office. He was unable to work for more than an hour without experiencing a headache and a burning, stinging sensation in the eyes. He had had ten refractions performed in six years and also had been given ocular muscle exercises. At the time of examination he was wearing the following lens combination:

Right eye: — 4.50 D. sph. ⊃ — 1.75 D. cyl., axis 85; 2½ degree prism base in and 2½ degree prism base up; resulting vision, 6/6.

Left eye: + 0.25 D. sph. ⊃ + 1.00 D. cyl., axis 60; resulting vision, 6/6.

Examination of the lids, conjunctivas, corneas and ocular fundi gave normal results. On downward gaze, while the patient was wearing his glasses, 3 D. of left hyperphoria was demonstrated. On phoropter examination in direct gaze the patient was found to have 2 D. of left hyperphoria in near and distant vision, 2 D. of exophoria in near vision and 1 D. of esophoria in distant vision. Refraction was done under homatropine cycloplegia, with the following result:

Right eye: — 3.00 D. sph. ⊃ — 1.75 D. cyl., axis 85; resulting vision, 6/5.

Left eye: + 1.50 D. sph. ⊃ + 1.00 D. cyl., axis 70; resulting vision, 6/5.

Two days later the postcycloplegic lens combination and prescription was:

Right eye: — 3.00 D. sph. ⊃ — 1.75 D. cyl., axis 80; 1 D. prism base up.

Left eye: — 1.00 D. sph. ⊃ + 1.00 D. cyl., axis 70; 1 D. prism base down.

After the patient had read for several hours wearing this prescription a left hyperphoria of 4 prism diopters was found on downward gaze. This was constant on repeated measurement, and the patient was given divided vertical prisms in the form of clip-on lenses: right, 2 prism diopters base up, and left, 2 prism diopters base down. He was advised to use these only while doing close work.

Two months later the symptoms of asthenopia had disappeared, and compensated prism bifocals were ordered which gave 4 prism diopters of compensation.

CASE 2.—A white man aged 35 complained of inability to perform close work without ocular fatigue and epiphora. He had had five refractions performed during the past three years, the last one four months before the present examination. He was employed as a telegrapher. On examination the lids were normal, the conjunctiva was hyperemic, the corneas were clear and the ocular fundi were normal. He was wearing the following lens combination:

Right eye: — 2.37 D. sph. ⊃ — 3.87 D. cyl., axis 163; resulting vision, 6/15.

Left eye: — 0.37 D. sph. ⊃ + 1.00 D. cyl., axis 102; resulting vision, 6/6 + 2.

Despite the poor vision of the right eye, fusion was present as demonstrated by the Maddox four dot test. On downward gaze at his particular working distance a left hyperphoria was demonstrated. Three diopters of vertical prism was required to bring the horizontal streak of the Maddox rod through the light. Examination of the phoropter in direct gaze showed esophoria of 1 prism diopter in near vision and of 3 prism diopters in distance. Hyperphoria was not demonstrated. Refraction was done under homatropine cycloplegia. At the postcycloplegic examination the patient accepted:

Right eye: — 1.50 D. sph. ⊃ — 3.25 D. cyl., axis 165; resulting vision, 6/15.

Left eye: — 0.25 D. sph. ⊃ + 1.25 D. cyl., axis 95; resulting vision, 6/6 — 1.

With this correction on downward gaze at his working position a 2 diopter prism was required to bring the streak of the Maddox rod through the light on

repeated examinations. Divided vertical prisms in clip-ons were given: right, 1 prism diopter base up, and left, 1 prism diopter base down. The patient was told to use these for close work. He was completely relieved of symptoms with them. A slab-off lens was given two months later, giving the equivalent of 2 prism diopters of compensation.

CONCLUSIONS

1. Many patients with moderate and high degrees of anisometropia are comfortable in near vision without prism compensation, and for these compensation is not indicated.

2. Others, however, usually bookkeepers and other persons doing much close work, have a definite need for compensating prisms and if they are not provided frequently suffer greatly from asthenopia, at times presenting the picture of an ocular neurosis.

3. The presence of poor vision in one eye does not obviate the necessity for compensation if binocular single vision is shown to be present by the usual tests.

4. We have found it more satisfactory to treat these conditions as one would a hyperphoria in downward gaze due to a muscle defect, that is, by repeated measurements with the Maddox rod at the individual working distance. The amount of prism necessary by this method is usually less than the amount indicated mathematically when 8 or 10 mm. is taken as the arbitrary amount of depression of the eyes in downward gaze.

5. It seems more logical and in our hands has been more satisfactory to decide on the amount of compensation necessary ourselves rather than to leave the matter to be taken care of arbitrarily by the optician.

TREATMENT OF GONORRHEAL DISEASES OF THE EYE WITH SULFANILAMIDE

THREE YEARS' CLINICAL EXPERIENCE

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Treatment of gonorrheal infection of the human eye has as its immediate objective the early eradication of the gonococcus and its toxins from the conjunctiva and from the superficial layers of the cornea. The speed of this successful attack will directly influence prevention of corneal complications. Gonorrheal conjunctivitis usually has complications, and involvement of the cornea is the most serious result when it does occur. It is usually less difficult to save the eye of a newborn child who has this disease than it is to save the adult eye, in which, heretofore, involvement of the cornea has usually led to some hopeless complication. Ophthalmia neonatorum is the cause of blindness in about 10 per cent of cases in Europe; in just what percentage blindness is due to gonorrheal infection it is impossible to state.

At the Philadelphia General Hospital, where more than 80 patients with gonorrheal infection of the eye have been treated in the last three years, 42 patients were treated with sulfanilamide, administered both locally and internally. These patients were from the ophthalmologic services of Drs. L. W. Deichler, A. Cowan and T. A. O'Brien and from my own service.

From this clinical experience there has been evolved what is now a standard routine for the treatment of this disease. The results have brought about a remarkably lessened incidence of corneal complications and a decrease in the duration of hospitalization; thus an economic and social problem in this community has been successfully solved. It is the belief of physicians in the ocular services at the Philadelphia General Hospital that if the plan of treatment now pursued there is followed rigidly gonorrheal infection of the human eye will be relegated to the category of the less serious ocular diseases.

In the present series of 42 patients treated with sulfanilamide, there were 14 infants with ophthalmia neonatorum of gonorrheal origin and 28 patients who had gonorrheal infection of the conjunctiva and cornea. All of the 14 infants were discharged without corneal involvement, but there were 5 patients with corneal complications on discharge among the 28 who had contracted the disease at a later time after birth.

On admission smears are taken of material from both eyes. Each patient is placed in a special isolation ward or cubicle. Nursing service is maintained for twenty-four hours in each case, under the supervision of a graduate nurse experienced in handling diseases of the eye. Smears of material from each conjunctival cul-de-sac are made at daily intervals; sometimes these smears are made twice each day. Treatment is instituted immediately on admission and continued under the direct supervision of the chief or one of his assistants until the patient has been approved (by either of them) for discharge.

The treatment now followed at the Philadelphia General Hospital is the use of sulfanilamide internally in a dose of 1 to 2 grains (0.06 to 0.12 Gm.) per pound of body weight, but never more than 100 grains (6 Gm.), for the first twenty-four hours of hospitalization, and irrigations of the involved eye with 2 per cent boric acid solution followed by the free instillation of an 0.8 per cent solution of sulfanilamide at ten minute intervals day and night.

The local use of this saturated solution of sulfanilamide is complemented by an adequate amount of internal treatment with the same drug. It has been reported by many physicians that the bactericidal effect of this drug is due primarily to its chemical reaction in the blood stream. It is my own belief that the internal treatment could be dispensed with in favor of the almost continuous irrigation of the involved cul-de-sac, with an equally good result. However, it is not within my province to conduct experiments of this nature when so many authorities have advocated the importance of internal use of this drug.

When sulfanilamide is given to infants, the prescribed amount is crushed and placed in water in a feeding bottle. No difficulty in its ingestion and passage through the nipple holes is encountered if the nurse agitates the bottle to keep the undissolved particles in suspension.

Studies of the blood are performed in all cases before use of the drug and during treatment. Toxic effects of sulfanilamide have not been encountered, although in the early stages administration of the drug was stopped or the dose decreased when there was lowering of the red blood cell count and the hemoglobin level.

After the first twenty-four hours of treatment the internal dose of the drug is decreased to ¼ to ½ (0.025 to 0.03 Gm.) for each pound of body weight, again depending on the particular case, and the local irrigations are continued at decreased intervals.

The use of a solution of atropine sulfate in strengths varying from 0.25 to 1 per cent according to the age of the patient, iced wet applications for ten minutes every hour and the use of a Bullar shield when only one eye is involved complete the therapeutic measures. The time-honored use of iced compresses and atropine sulfate solution needs no endorsement of its immediate and far reaching effects in attacking this

infection and its complications. Atropine sulfate is instilled until discharge, and iced compresses are used with a frequency that is indicated clinically.

No patient is considered ready for discharge from the hospital until negative results of examination have been reported on three successive days by the head of the clinical laboratory and there are no sequelae or complications which need further treatment.

In many of the cases of early involvement variable periods elapsed before the first negative smear was obtained and particularly the third successive negative smear. However, it might be important to note at this time that in the last 6 cases observed the following numbers of days of hospitalization were required to obtain three successive daily negative smears: four days, five days, three days, three days, three days and six days respectively. From these figures it can be noted that 1 patient received treatment but one day before the first of three successive negative smears was obtained. The second received treatment for two days. From each of the next 3 a negative smear was obtained within the day when treatment was started. The last patient observed had three days of treatment before the first of the three successive negative smears was obtained.

Complications which were present either on admission or during the course of treatment were observed. They included rupture of the cornea with prolapse of the iris on admission; posterior synechiae, possibly from old iritis; corneal ulcer present on admission (2 cases), and leukoma present on admission (1 case). One patient came in with superficial erosion of the cornea, which was clear on discharge. Nine were admitted with hazy corneas and discharged with clear corneas. One patient died three days after admission, on the day after the first negative smear was obtained. The patient with corneal rupture and prolapse of the iris was discharged, and all trace of this patient was lost.

At this time the ophthalmic surgeons at the Philadelphia General Hospital are encouraged by the results of treatment of gonorrheal infection of the eyes by local and internal use of sulfanilamide. The first patient treated by this method was in Dr. O'Brien's service (July 1937). The results in the first 6 cases were reported by me before the Eye Section of the Philadelphia County Medical Society on March 3, 1938.

After three years of clinical trial and observations, the method of treatment has evolved inself into almost a constant series of treatments during the first twenty-four hours. Even a meal or a feeding is not allowed to be completed without an interruption for irrigation and instillation of solution of sulfanilamide. In the second twenty-four hours the treatment is less constant, and with the appearance of negative smears and the absence of clinical signs of corneal involvement the dose is decreased. The nurses in charge of the patients cannot be too warmly

complimented for the success which has been attained. Any method of approach to the very serious problem of conjunctival infection which decreases the incidence and possibility of corneal involvement justifies the effort and means expended.

The increase in public knowledge aimed at early treatment of genital gonorrheal infections will be no small factor in helping to eliminate infection appearing in the eye. The early arrest of gonorrhea by new and improved methods of treatment also will help to keep at a minimum the incidence of this type of complication.

A variable number of days elapsed between the onset and the inauguration of treatment. In 1 case two and one-half months was given as the time during which a puslike discharge from the eye had been present; the condition was diagnosed on the patient's admission to the hospital as gonorrheal infection of the eye. The patient was admitted with a steamy cornea and discharged with a clear cornea.

Among other infections of long duration were included 1 that had been present for twenty-six days, 1 present for seventeen days and others present for fifteen, fourteen and eleven days respectively. In general, the average interval between onset of the condition and admission to the hospital was from two to three days. Many of the patients who were infected and who had had symptoms for longer than two or three days before admission presented no unusual corneal signs when first examined at the hospital.

At present there has been no decrease in the average number of patients admitted each year, but there has been a remarkable decrease in the number of patients discharged with involvement of the cornea.

UVEOPAROTITIS (HEERFORDT)

A NOTE ON PROGNOSIS

SAMUEL I. KAUFMAN, M.D.
CHICAGO

Uveoparotitis, a syndrome first described by Heerfordt in 1909 under the name of "febris uveoparotidea subchronica," regarding the cause of which there is still a good deal of diversity of opinion, has been receiving increasing attention of late. A number of articles have appeared in the United States and abroad, among them several containing exhaustive bibliographies.[1] It would appear from the number of case reports that uveoparotitis is a relatively rare condition. Folger,[1a] for instance, found mention of only 60 cases in the entire literature up to 1936. Undoubtedly there are cases which are not being reported or which because of the atypical appearance of the condition escape recognition. Yet this condition must indeed be infrequent if several ophthamologists of my acquaintance, of large clinical experience, have never encountered a case in their practice. The ophthalmologist who is confronted with this condition, perhaps for the first time, may take comfort in the knowledge that the uveitis accompanying this syndrome, in spite of its protracted and stormy course, shares in the well marked tendency to "spontaneous recovery" [2] which seems to characterize this disease. This observation is further confirmed by the case report presented here. This is all the more gratifying in view of the unknown cause and the resulting sole dependence on symptomatic treatment.

REPORT OF A CASE

Present Illness.—E. S., a white married woman aged 22, suddenly had Bell's palsy on the right side and at the same time noted a swelling in the left parotid area on June 2, 1938. Four days later both eyes became somewhat painful and reddened; tearing and photophobia were noted. In about two weeks the palsy cleared up, but by this time a similar palsy had developed on the left side. Since the onset of this illness the patient had also noted dryness of the mouth, some loss of the sense of taste, a rash around the ankles, a crackling sensation in the mandibular joints, pain in the left shoulder blade and a feeling of general malaise.

1. (*a*) Folger, H. P.: Uveoparotitis (Heerfordt): Report of Case, Arch. Ophth. **15**:1098 (June) 1936. (*b*) Walsh, F. B.: Ocular Importance of Sarcoid: Its Relation to Uveoparotid Fever, ibid. **21**:421 (March) 1939.

2. Garland, H. G., and Thomson, J. G.: Uveoparotid Tuberculosis, Quart. J. Med. **2**:157, 1933.

Family and Past Histories.—The patient first came under my attention on June 27, 1935, complaining of "weak eye muscles" and twitching of the lids. On examination the external appearance, ocular movements, conjunctivas, pupils, media, tactile tension and fundi were found to be normal. Under homatropine cycloplegia the following correction was ordered:

Right eye: —0.25 D. sph. —1.00 D. cyl., axis 90; resulting vision, 1.0
Left eye: —0.75 D. sph. —0.75 D. cyl., axis 90; resulting vision, 1.0

The patient was not seen again by me or any other oculist until the onset of the present illness. There was no history of tuberculosis; no symptom of this condition was present, and the patient had had no contact with the disease. There had been no surgical operations or accidents.

There was no family history of disease similar to the present illness, diabetes, carcinoma, pulmonary disease or renal disease.

Marital Status.—The patient had been married one year. Her husband was living and well. She had no children and had not been pregnant.

Ocular Examination (June 2, 1938).—Vision in the right eye was 20/50 and in the left eye 20/30 with the correction given on June 27, 1935. No change in the present correction was indicated. Externally there were some blepharospasm and a faint ciliary flush, a very mild reaction considering the massive exudation in the anterior segment of the eye. The posterior surface of the cornea was studded with lardaceous deposits of all shapes and sizes. The deposits were eventually so numerous that they tended to coalesce to form massive blotches which could be seen through the cornea without magnification, with the naked eye. The pupils dilated readily under the influence of atropine sulfate, uncovering two synechiae in the right eye which eventually disappeared. The irides contained several discrete, golden yellow, sharply circumscribed nodules midway between the pupillary and ciliary margins unlike those seen in tuberculous iritis. The vitreous in both eyes at this time contained fine opacities producing a diffuse haziness which was more pronounced in the right eye than in the left. The tension was normal in both eyes. The retinal vessels were engorged, but the optic disks were of normal appearance. Peripherally and centrally the visual fields were normal. Both lacrimal glands were enlarged.

Physical Examination of Affected Parts.—A nodular, indurated mass was felt over the ramus of the left mandible and extended over the masseteric region, along the course of Stensen's duct. A similar mass of lesser extent was palpable in the right masseteric region. The openings of Stensen's ducts did not appear inflamed. The facial weakness involved the forehead, the lids and the mouth. The lymph nodes in the neck were not enlarged. The palate was normal on phonation, and the voice was unimpaired. Taste sensation for quinine, salt and sugar was normal in the anterior two thirds of the tongue. (The patient maintained, however, that the sensation of taste was definitely dulled.)

Laboratory Data.—The Kahn and Wassermann reactions of the blood were negative. The reactions to Mantoux tests were repeatedly negative. The erythrocytes numbered 4,500,000 per cubic millimeter of blood and the leukocytes 5,400, with neutrophils 75 per cent, lymphocytes 23 per cent and monocytes 2 per cent. No immature cells were noted. The blood pressure was 120 systolic and 80 diastolic. The value for blood sugar was 82 mg. and the nonprotein nitrogen 27 mg. per hundred cubic centimeters. The urine contained no albumin or sugar. Agglutination tests for typhoid, dysentery, paratyphoid, Brucella abortus infection

and Bacillus tularensis infection gave negative results. Roentgen examination of the chest showed general enlargement of the hilar glands. The lung parenchyma showed no abnormal markings. The nasal sinuses were normal.

Clinical Course.—There was a low grade fever throughout the course, the temperature ranging between 97.2 and 100. The disease followed a chronic course for over a year, reaching its greatest intensity within the first six months. None of the remedies used seemed to produce any striking change in the progress of the condition. During its height the media were so cloudy that no details of the fundi could be made out in spite of the wide dilatation of the pupils. Gradually, however, the media began to clear; the deposits on the cornea, the iris and the anterior capsule of the lens began to disappear, and vision gradually improved. On July 12, 1939, vision in the right eye was 20/30, and in the left eye, 20/20. A few discrete deposits were still to be observed on the posterior surface of the cornea and a few opacities in the vitreous of the right eye.

Treatment included the use of atropine sulfate, ethylmorphine hydrochloride, local heat, hyperthermic treatments with a temperature of 105 F. maintained for five hours, roentgen treatment over the eyes and both parotid regions, typhoid vaccine given intravenously and several courses of sulfanilamide.

COMMENT

Every form of exudative uveitis, especially if bilateral, is of great concern to the ophthalmologist and of grave import to the patient. The longer the process lasts unabated, the more certain are the sequelae, which will endanger the sight. It is therefore heartening when confronted by a disease of unknown cause to know that the chances for eventual recovery are favorable.

BLACKBERRY THORN IN THE ANTERIOR CHAMBER OF THE EYE FOR TWELVE YEARS

EDWARD C. ALBERS, M.D.
CHAMPAIGN, ILL.

It is well known that an intraocular foreign body can be tolerated for a considerable length of time. Much experimental and clinical research has been done on this subject, and it has been found that the reaction of the eye to such an accident depends on a number of factors. The factors determining tolerance are: (1) the septic or aseptic nature of the foreign body; (2) the type of organism carried into the eye; (3) the ocular tissue involved; (4) the chemical composition of the substance, and (5) the mechanical irritation produced.

Retained intraocular foreign bodies which are inert may remain free in the eyeball or become encapsulated by organized exudate, the reactions depending entirely on the situation of the foreign body. Violent inflammation may occur at the time of the accident even though no infection can be demonstrated, or reactions may occur many years after the accident

The following case is presented to illustrate how long an inert thorn may remain in an eye without causing any reaction after the initial reaction has subsided.

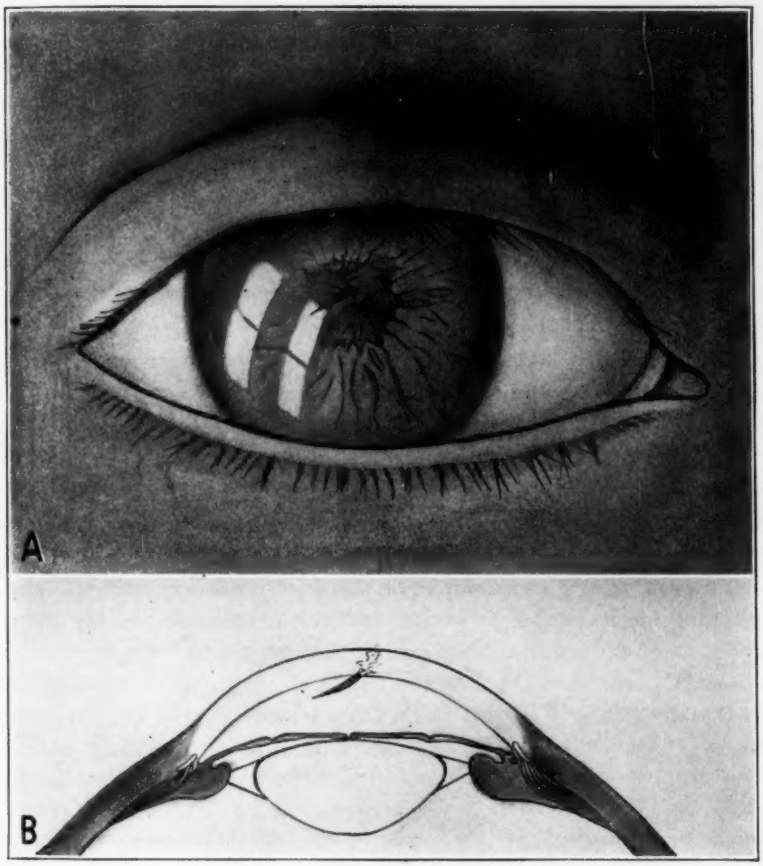
REPORT OF A CASE

A white man aged 50 came to the Christie Clinic on April 22, 1940 for an ocular examination. He stated that his right eye had been injured while he was cutting blackberry bushes twelve years before. He picked up a blackberry bush, and a branch accidentally struck the eye; so he went to his family physician immediately and was told that he had a blackberry thorn in his right cornea. He said that the physician removed it, but the next day the eye became red. After a stormy bout of inflammation and intense pain, which lasted for one month, the eye became blind. Since that time he had been able to see only light with that eye, and there had never been any recurrence of the pain.

Examination revealed the following data: Light perception and projection were good. The lids and conjunctiva were normal. There was a white scar just nasal to the apex of the cornea, 1 by 1 mm. in diameter, slanted laterally and extending through its entire thickness. The base of a glistening brown tapering

From the Department of Ophthalmology, the Christie Clinic.

lunate thorn 4 mm. long was attached by a small amount of fibrous tissue to the posterior aspect of the scar at Descemet's membrane. The thorn extended laterally and slightly downward, was completely surrounded by aqueous and did not touch the iris or the lens. The anterior chamber was a little deeper than normal. The iris was the same color as its fellow and did not show any evidence of active



A, anterior view of the right eye. *B*, diagrammatic appearance of the right eye in horizontal section, showing the thorn.

inflammation. The pupil was very small and was bound down to a cataractous lens by posterior synechiae; so the fundus could not be seen. The tension measured 18 mm. of mercury (Schiötz).

The history of the left eye was irrelevant, and examination of it revealed no abnormality.

THE SUPERNUMERARY CARUNCLE

REPORT OF A CASE

BENJAMIN FRIEDMAN, M.D.
NEW YORK

The rarity of a supernumerary caruncle prompted Stephenson,[1] who is credited with the first description of the condition (1896), to make the following remark: ". . . I was unaware that the caruncle, like the tragus, mamma, or nipple, was subject to reduplication." The second case on record is that of Schreiber[2] (1913), and then follow 2 cases reported by Feigenbaum[3] (1931). The present communication is written with the intention of adding a fifth case, in which there was a double reduplication.

The characteristic location of the supernumerary caruncle is just behind the inferior punctum, on the tarsal conjunctiva. It thus lies close to the normal caruncle but is not connected with it. Grossly the tissue possesses a somewhat granular surface, with the yellowish subepithelial mottling which is conspicuous in the natural caruncle. Lanugo hairs may be readily observed. The mass is not quite as large as the parent caruncle and may be mistaken for a granulating chalazion.

The embryologic explanation of the supernumerary caruncle makes interesting speculation. The normal caruncle originates from the extreme nasal part of the embryonic lower lid and begins to be seen in the 35 mm. stage (Ask[4]). The lower canaliculus originally extends laterally into the substance of the lower lid considerably farther than it does at birth; as it moves inward the portion of the lower lid which it embraces in its arc becomes the anlage of the caruncle. This tissue becomes isolated and pushed farther toward the nose, to its ultimate position. The caruncle thus comes to contain in rudimentary form all the structures which are indigenous to the lids—sebaceous glands, hair follicles, hairs, a modified form of Krause's glands, muscle fibers, connective tissue and fat.

Feigenbaum attributed to Ask the theory that in cases of supernumerary caruncle the lower canaliculus is forked early in its develop-

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From the service of Dr. Bernard Samuels, New York Eye and Ear Infirmary.

1. Stephenson, S.: Ophth. Rev. **15**:8, 1896.
2. Schreiber, L.: Arch. f. Ophth. **84**:431, 1913.
3. Feigenbaum, A.: Klin. Monatsbl. f. Augenh. **87**:760, 1931.
4. Ask, F.: Anat. Anz. **30**:197, 1907; Anat. Hefte **36**:255, 1908.

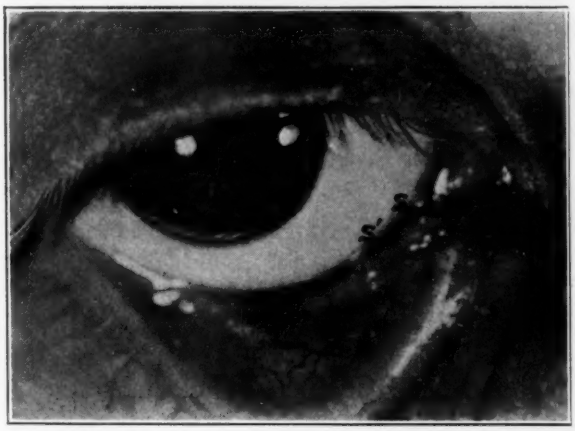


Fig. 1.—Double supernumerary caruncle in the right lower lid. *C*, normal caruncle; *S* and *S'* supernumerary caruncles.

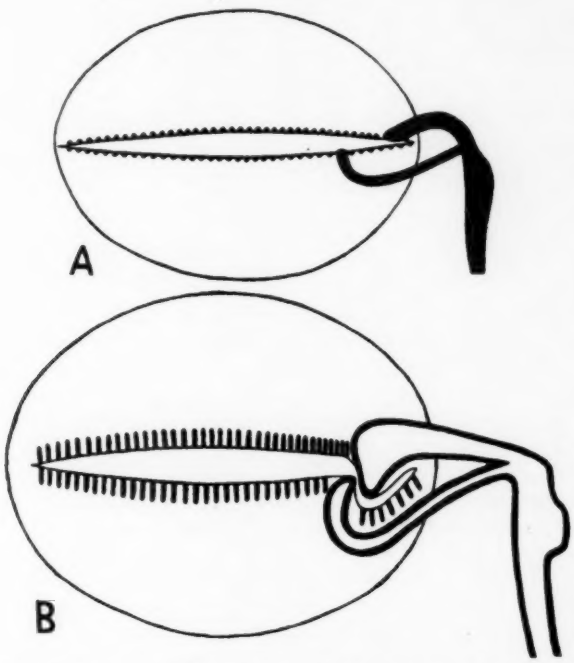


Fig. 2 (after Ask [4]).—*A*, eye of 40 mm. embryo. The drawing is a diagrammatic representation of the position of the inferior canaliculus in early embryonic life. *B*, eye of 170 mm. embryo, showing the nasalward migration of the inferior canaliculus. Note the sequestrum of lower lid tissue which is presumably snared off to form the caruncle.

ment, thus snaring off two caruncles instead of one. This does not explain why the lower punctum and canaliculus fail to become reduplicated.

It seems to me that the lower canaliculus is not the crucial determining agent in the production of the supernumerary caruncle or even the natural caruncle. There is a recognizable condensation of the caruncle anlage at a very early stage, and the tissue appears destined in its own right to grow into a caruncle even without the intervention of the canaliculus. Proof of this is found in instances of congenital absence of the punctum and canaliculus; the caruncle is found to develop normally in these cases. Furthermore, in those cases in which there is a reduplication of the punctum and canaliculus the caruncle does not share in the reduplication. I believe in cases of supernumerary caruncle the original caruncular tissue starts growth as two independent masses on either side of the intruding canaliculus; the nasal part moves along as it should with the canaliculus, while the temporal portion remains behind to fulfil its destiny in the lower lid.

The condition in the case to be reported was distinguished by a double reduplication in the form of two lobulated masses, one just behind the lower punctum and the other more temporally placed. In the cases which preceded mine one might postulate the reduplication to be the result of the caruncular anlage being split in two by a normal lower canaliculus, but such a theory would not explain in my patient the presence of two distinct supernumerary masses. It seems more likely that the caruncular anlage was originally laid down in three separate islands, of which only one developed naturally.

REPORT OF CASE

Mr. J. K., aged 46, presented himself at the clinic for glasses to assist him in his reading. Examination of the right lower lid disclosed two small lobular masses lying side by side on the inner surface. The growths were well below the level of the lid margin and were invisible while the lid was in its normal position. The nasal mass was situated just under the lower punctum; the other was connected with it by a narrow bridge of tissue. Except for this point of fusion, the lobules were quite free of each other. They were about 3 mm. distant from the caruncle and independent of it. The growths were fixed to the underlying tarus by a small base, but the major portions of the masses were freely movable. The masses were flattened and measured about 3.5 mm. in diameter and about 2.5 mm. in thickness. Comparison with the normal caruncle readily disclosed a similarity in tissue structure. Closer observation with the slit lamp revealed a single lanugo hair in the nasal mass and the openings of seven or eight hair follicles on the surface of both lobes. A small oil cyst was noted in the temporal mass.

The growth was easily removed, and microscopic study substantiated the clinical diagnosis. The left caruncle was not reduplicated.

Histologic Examination.—The surface covering consisted of a layer of stratified cylindric epithelium from three to eight cells in thickness. A large number of mucous (goblet) cells were to be seen. Many diverticula of Henle were present.

Groups of lymphocytes and plasma cells as well as individual cells were distributed throughout the loose subepithelial connective tissue. A great part of the tissue consisted of large sebaceous glands, partly surrounded by round cells. Bands of striated muscle were seen in the superficial and deep layers. Several hair follicles were evident. Deep within the tissue were to be seen small groups of lobules of a gland resembling the lacrimal gland; these were considered analogous to the glands of Krause. Some sections contained remnants of fat tissue.

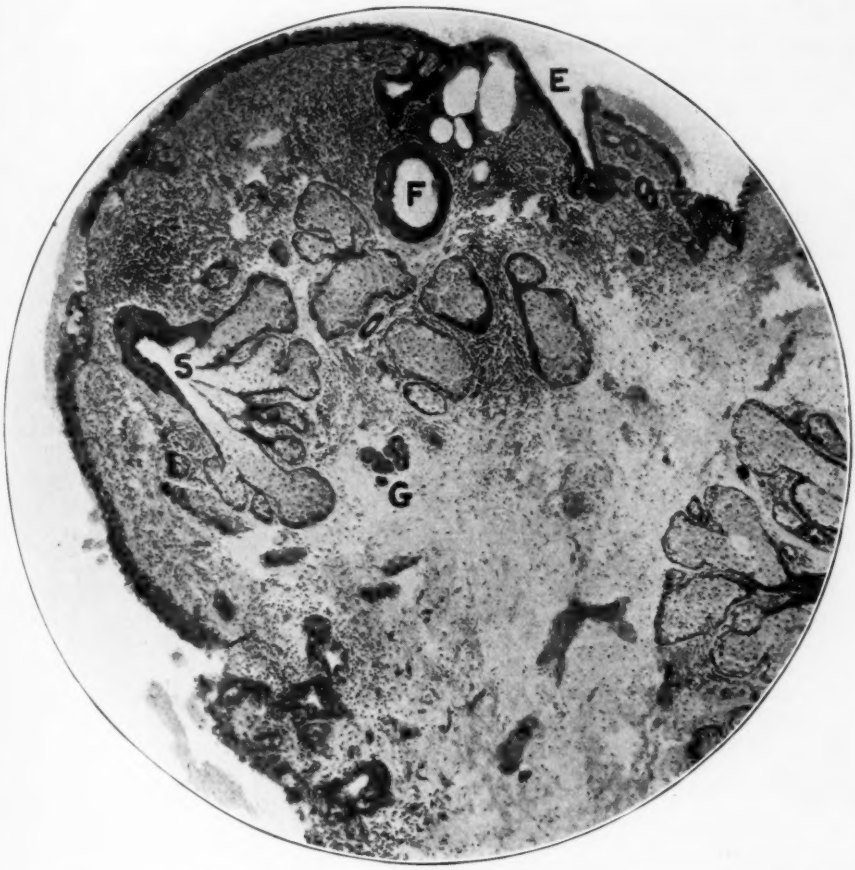


Fig. 3.—Photomicrograph of a section through a supernumerary caruncle. *E*, columnar epithelium with diverticula of Henle. Note the underlying round cell infiltration. *F*, hair follicle. *S*, sebaceous glands. *G*, gland similar in appearance to the lacrimal gland.

The observations corresponded, therefore, to the histologic picture of the normal caruncle except for the absence of stratified squamous epithelium. The presence of lymphocytes did not indicate chronic inflammation, since clusters of lymphocytes are found in normal caruncular tissue.

It is interesting to note that in this case the epithelial covering of the misplaced caruncular masses consisted of columnar cells, simulating the cell shape of the lid conjunctiva rather than the normal caruncular epithelium. Stephenson stated that the tissue in his case consisted of an "epidermic and a dermic portion." Schreiber was even less informative. Feigenbaum's tumors were covered by flattened epithelial (evidently squamous) cells. Peschel [5] reported an instance in which the caruncle was in its natural position but was surrounded and covered by epidermis and was connected with the lower lid by a strip of epidermis. Such an abnormality finds its counterpart in the normal caruncular anatomy of certain animals.[6]

5. Peschel, M.: Zentralbl. f. prakt. Augenh. **27**:148, 1903.

6. Duke-Elder, W. S.: Text Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1932, vol. 1, p. 230.

Clinical Notes

AN IMPROVED BINOCULAR GONIOSCOPIC APPARATUS

DONALD WEEKS BOGART, M.D., NEW YORK

In a previous publication [1] I described in detail a most satisfactory binocular gonioscopic apparatus as presented before the Section of Ophthalmology of the New York Academy of Medicine.

Two more years of experience with this device has definitely proved its worth but has altered some of my original ideas. Certain changes have been introduced to enlarge the scope of usefulness of the gonioscopic apparatus and it is for this reason that I again bring it to the attention of ophthalmologists.

I now believe that a satisfactory examination of the angle of the anterior chamber may be quickly done with a contact lens, a magnifying binocular loupe and a powerful, small beam flashlight. It is, however, of the utmost importance to have on hand a more powerful binocular apparatus to study in detail certain aspects not apparent under very low power, and it is for this purpose that I employ the apparatus previously described.

Figure 1 shows in detail the suspension of the binocular slit lamp microscope and the added Barkan lamp for illumination of my original apparatus. It is to be noted that in this illustration a simple ball and socket tripod head is utilized for motion. A large panoramic tripod head with all motion controlled by the action of a heavy lever has been added. This has been inserted in place of or (as illustrated) in conjunction with the ball and socket tripod head. It markedly increases the usefulness and ease of manipulation of the apparatus. The particular tripod head that I have used is made by the Albert Specialty Company, of Chicago, and comes as part of the "Royal" tripod.

The working parts exclusive of the panoramic tripod head are shown in figure 2. The Barkan lamp with the adapting rod attached, the ball and socket tripod head, the rod to adapt this to the copying stand and the binocular slit lamp microscope head with its base drilled to accept the screw of the tripod head are shown from the upper left, reading counterclockwise.

It is obvious that this may be quickly slipped into place with the base under the patient's head (the contact lens being retained from the simple examination) and centered for full detailed examination of any portion of the anterior chamber. One may utilize a variety of powers and one has at all times adequate illumination.

1. Bogart, D. W.: A New Binocular Gonioscopic Apparatus, Arch. Ophth. **21**:709 (April) 1939.

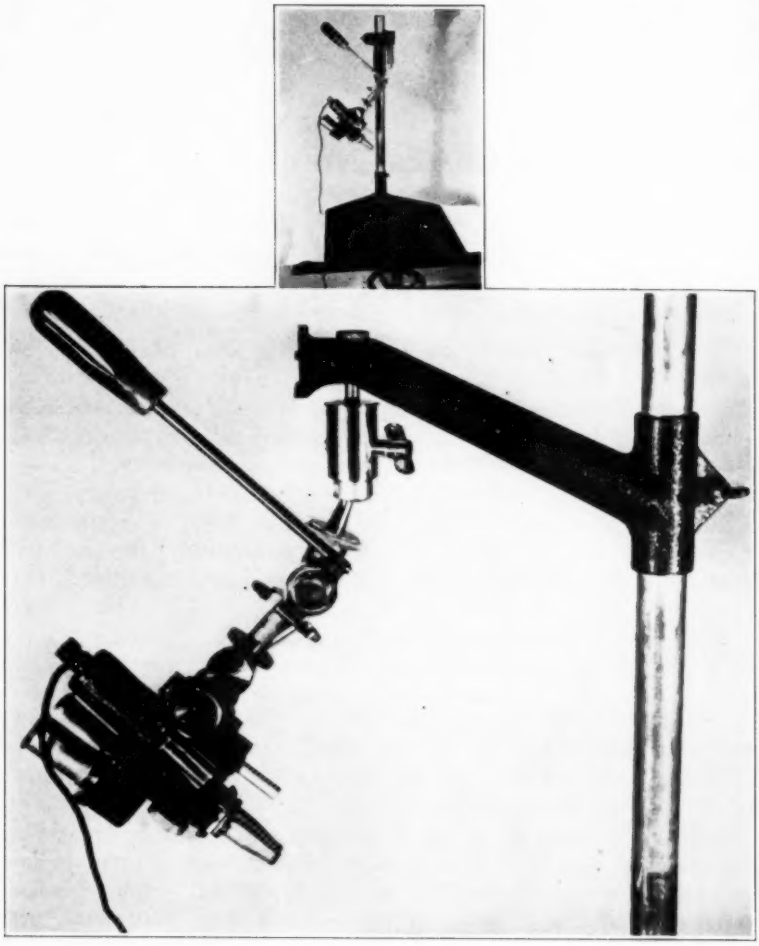


Fig. 1.—Improved gonioscopic apparatus with lever control of motion.

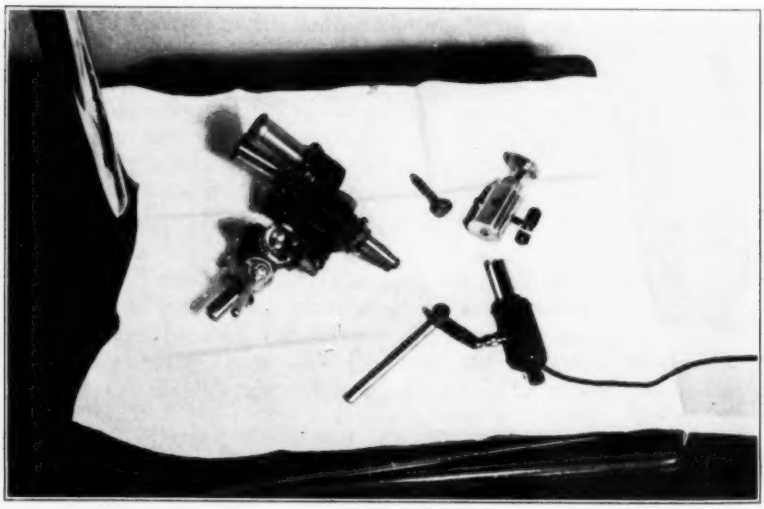


Fig. 2.—Working parts exclusive of the panoramic tripod head.

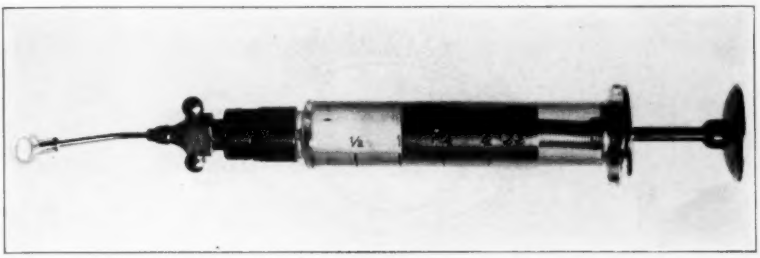
Photomicrographs are easily obtained merely by adding a device to one of the eye pieces of the microscope. The device which I employ is the Leitz "Micro Ibso" attachment, to which a Leica camera is attached. The camera may be focused by means of a side eye piece, and at precisely the right instant the photograph may be taken.

20 East Fifty-Third Street.

A NEW TIP FOR DIMITRY'S SUCTION SYRINGE

EDMOND L. COOPER, M.D., DETROIT

The suction syringe introduced by Dimitry [1] in 1939 has rendered the intracapsular extraction of certain types of cataract much easier and less liable to complications. I have used Dimitry's apparatus with considerable success, especially in cases of hard cataracts with large nuclei and hypermature cataracts when the capsule is tense and a



Transparent suction cup attached to Dimitry's syringe.

forceps tends to slip. A report of the cases in which Dimitry's syringe was used is being compiled and will be published later.

The purpose of this paper is to present a new suction cup for use on Dimitry's syringe. His instrument as originally designed provides a small needle of platinum or gold on the end of which is a flat discoid cup of sheet gold. When this is introduced into the anterior chamber and the previously depressed plunger is released the lens is sucked firmly against the cup and held there. However, when the suction cup is in place on the lens it is large enough to occupy the pupillary space completely, and the lens is thus hidden from view.

Because of this I had made for me by V. Mueller and Co., of Chicago, the makers of Dimitry's instrument, a suction cup of a transparent plastic material. The illustration shows this transparent suction cup attached to Dimitry's syringe. It is made as near as possible to the original specifications. It has the advantage over a gold suction cup of allowing the lens to be visible while it is being grasped and extracted.

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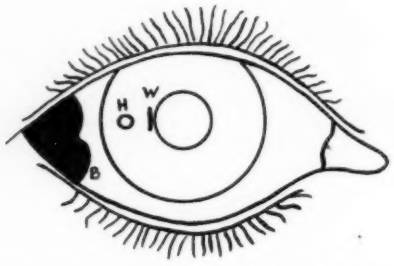
1. Dimitry, T. J.: Evolution of a Sucking Disk for Intracapsular Extraction of Cataract, Arch. Ophth. **21**:261 (Feb.) 1939.

SUBCONJUNCTIVAL HEMORRHAGE AS A SIGN OF DOUBLE PERFORATION OF THE GLOBE BY A FOREIGN BODY

BENJAMIN FRIEDMAN, M.D., NEW YORK

I wish to reiterate an observation which I made a number of years ago, when I called attention to the occurrence of a subconjunctival hemorrhage in the presence of a penetrating foreign body as evidence of double perforation of the globe.[1] This occurs when the foreign body enters the cornea obliquely and leaves the eye through the sclera, at some distance from the point of entrance, to embed itself under the overlying conjunctiva. I stated:

> When the foreign body penetrates the eye by way of the cornea the passage is through an avascular stratum, and consequently there is no subconjunctival bleeding. The foreign body flies at such a high rate of speed that penetration usually occurs with but little mechanical shock to the globe as a whole; there



Drawing to illustrate the presence of subconjunctival hemorrhage in a case of double perforation of the globe. *W* indicates the point of entrance through the cornea; *H*, the hole in the iris; *B*, the subconjunctival hemorrhage. Note that the hemorrhage lies in the direction of flight taken by the foreign body.

> are therefore no hemorrhages under the conjunctiva such as are found in cases of contusion. The bleeding that takes place is usually within the globe, from direct injury to the intraocular vascular tissues.

When the sclera is perforated in exit bleeding takes place under the conjunctiva because of lacerations of the vessels of the choroid, the episcleral layers or the conjunctiva.

The simultaneous existence of a fresh perforating wound in the cornea and a distant subconjunctival hemorrhage should excite suspicion that the foreign body is outside rather than inside the globe and that it lies underneath the hemorrhagic spot. In such an instance a weak magnet ought to be held directly over the area of bleeding; a bulge at this site indicates the advisability of direct removal of the fragment through the conjunctiva, precluding the risk of a more vigorous pro-

1. Friedman, B.: Subconjunctival Hemorrhage in Intra-Ocular Foreign Bodies, Arch. Ophth. **6**:919 (Dec.) 1931.

cedure by which one might inadvertently reintroduce the foreign particle into the eye. Such a complication would be brought about if an attempt were made with the giant magnet to draw the metallic fragment out through the corneal entrance wound.

A case which illustrates further the localizing value of a subconjunctival hemorrhage in cases of double perforation of the eye was observed in the service of Dr. Bernard Samuels at the New York Eye and Ear Infirmary. A wound was present in the right cornea just over the temporal edge of the pupil, and a hole was evident in the iris at the same level but halfway between the pupillary border and the temporal limbus. A large subconjunctival hemorrhage began at the outer canthus, filling the upper and lower fornices as well as the epibulbar space, and extended almost but not quite up to the cornea. Roentgen examination located the foreign body outside the globe. But corroborative proof from the roentgenologist was really unnecessary, for no other explanation could logically have accounted for the subconjunctival extravasation of blood.

This is a sign worth remembering when accurate roentgen localization is unattainable or when the foreign body lies so close to the globe that the roentgenologist is uncertain whether it is inside or outside the eye. Of course, the further back the exit point is placed the more profuse must be the bleeding in order to become visible under the conjunctiva.

Ophthalmologic Reviews

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GONIOSCOPY AND GLAUCOMA

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Recent interest in gonioscopy is evident from the presentation of three papers and a lecture course on this subject during the 1940 meetings of the American Academy of Ophthalmology and Otolaryngology and the Pan-American Congress of Ophthalmology in Cleveland. Its value in clinical ophthalmology has been established by Troncoso, Thorburn, Werner, Vannas, Barkan and others. In the study of glaucoma it is valuable in several particular ways: first, in indicating the best location for surgical drainage in cases in which synechiae are present; second, in determining the prognosis and ascertaining the causes of operative failure; third, in indicating the type of secondary operation when reoperation is necessary; fourth, in the differential diagnosis between primary glaucoma and certain types of secondary glaucoma; fifth, in measuring the depth of the chamber angle (when gonioscopic observation is made with a microscope containing a micrometer eye piece), and last, but not least, in leading to a better understanding of the problem of glaucoma by way of a relatively new approach.

Aside from glaucoma, gonioscopic technic is helpful in studying certain pathologic changes in the area of the limbus and the chamber angle. Among these are anomalies (hydrophthalmos; congenital megalocornea; congenital coloboma; aniridia), tumors, cysts, foreign bodies and traumatic and inflammatory lesions.

In a previous paper [1] I presented the gonioscopic data collected in the glaucoma clinic of the Illinois Eye and Ear Infirmary during 1939. Those observations and statistics have been revised to include the new material of the past year and the follow-up findings on the old. The patients were observed gonioscopically for periods varying from two years to two months.

From the Illinois Eye and Ear Infirmary.

Aided by a grant from The Kellogg Foundation, Battle Creek, Mich.

1. Sugar, H. S.: Concerning the Chamber Angle: I. Gonioscopy, Am. J. Ophth. **23**:853, 1940.

Since Trantas'[2] observations of the chamber angle by direct and indirect ophthalmoscopic examination in 1902, all investigators have used contact lenses of various types to make the angle visible. Salzmann[3] used Fick's keratoconus contact lens and later designed one of his own. Koeppe[4] devised a lens with which he made slit lamp examinations of the angle. This lens is still in use and is the model after which Barkan's plastic contact lens is made.

Modern gonioscopy began in 1925, with a paper by Troncoso[5] in which be described the gonioscope and the "C" lens which he had suggested to Koeppe. He pointed out the important fact that synechiae are the result and not the cause of glaucoma. Together with Castroviejo,[6] he did important work on the comparative anatomy of the angle. His subsequent papers on gonioscopy have established his place as an authority in this field.

After Barkan[7] introduced his operation of goniotrabeculotomy in 1936, the need for better knowledge of the anatomy of the chamber angle caused a surge of interest in gonioscopy. As a consequence, the use of the gonioscope is becoming wider, not only in the large clinics but in the private offices of ophthalmologists throughout the United States.

GONIOSCOPIC APPEARANCE OF THE NORMAL CHAMBER ANGLE

The chamber angle consists of the angular space bounded by the iris root, the anteromedial surface of the ciliary body and the scleral wall. The histologic descriptions of the chamber angle in the literature have been confusing, particularly because the essential differences between the angles of lower animals and those of the human being and the primate have not been fully appreciated. These differences have been well described by Troncoso and Castroviejo.[6]

In embryonic life the space which later corresponds to the angle is filled by a mesodermal reticulum which, after partially atrophying to form the anterior chamber, gives rise to the iris stroma and the "meshwork of the chamber angle." The meshwork is divided into two por-

2. Trantas, A.: Ophtalmoscopie de la région ciliaire et rétrociliaire, Arch. d'opht. **27**:589, 1907.

3. Salzmann, M.: Die Ophthalmoskopie der Kemmerbucht, Klin. Monatsbl. f. Augenh. **52**:561, 1914.

4. Koeppe, L.: Das stereomikroskopische Bild des lebenden Kammerwinkels an der Nernstspaltlampe beim Glaucom, Klin. Monatsbl. f. Augenh. **65**:389, 1920.

5. Troncoso, M. U.: Gonioscopy and Its Clinical Applications, Am. J. Ophth. **8**:433, 1925.

6. Troncoso, M. U., and Castroviejo, R.: Microanatomy of the Eye with the Slitlamp Microscope, Am. J. Ophth. **19**:1, 1936.

7. Barkan, O.: A New Operation for Chronic Glaucoma, Am. J. Ophth. **19**: 951, 1936.

tions, scleral and uveal. The major portion is scleral and is known as the corneoscleral trabecula. Particularly in its posterior two thirds, it contains many spaces which have direct access to the aqueous. The uveal portion, the uveal meshwork, corresponds to the ligamentum pectinatum of lower animals. It consists of meridianally situated trabeculae which continue from the anterior border layer of the iris, line the angle recess and the ciliary body and usually extend onto the corneoscleral trabecula. Most of the trabeculae are very delicate and are only one or two cell layers in thickness, while some are comparatively thick and stand out against their background. The latter type, known as iris processes (fig. 1), may line the wall of the angle as

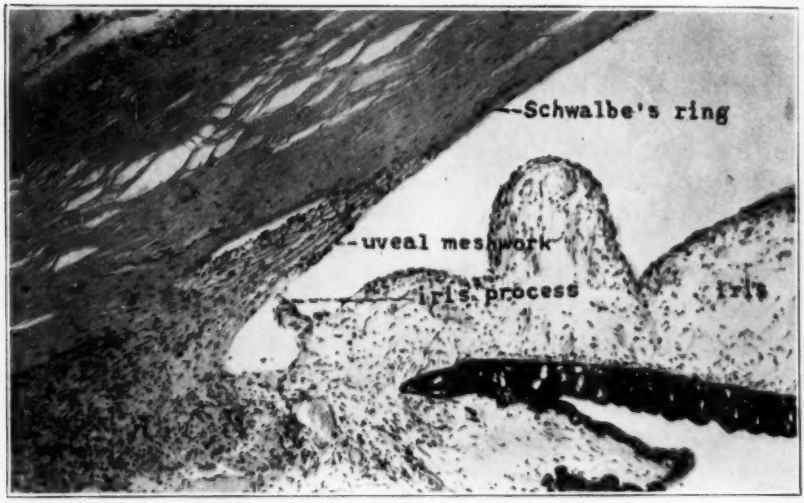


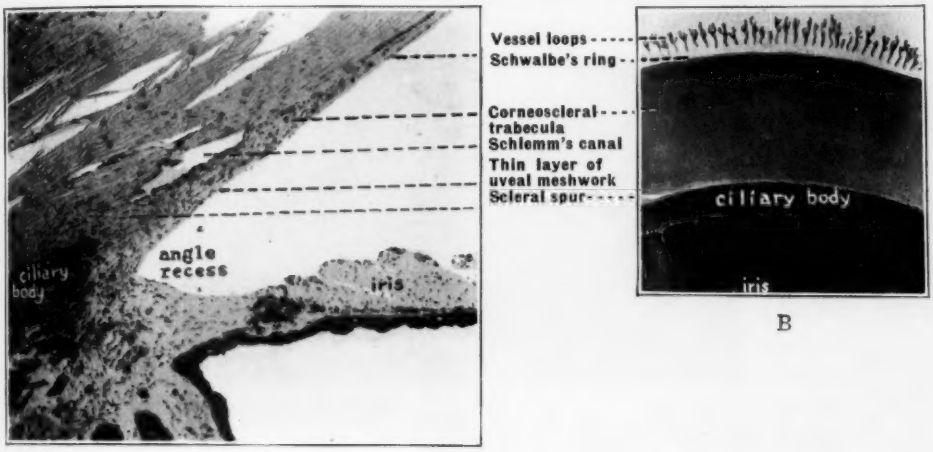
Fig. 1.—Section of an atropinized eye, showing the bridging type of iris process and a portion of the uveal meshwork on the trabecula.

described, or they may extend from the iris to the ciliary body or the trabecula, bridging the angle recess. The distinction between the bridging and the nonbridging type of iris processes is only a histologic one and cannot usually be observed gonioscopically. It would be best to use the term ligamentum pectinatum for the iris processes and the remaining uveal meshwork instead of for the corneoscleral trabecula or the entire angle tissue, as has been erroneously done in much of the literature. Similarly, the trabecular spaces have been erroneously alluded to as spaces of Fontana. Spaces of Fontana are not present in the human eye unless one uses the term for the spaces behind bridging iris processes as seen in tissue sections. Anderson [8] suggested that the term liga-

8. Anderson, R.: Hydrophthalmia or Congenital Glaucoma, London, Cambridge University Press, 1939.

mentum pectinatum be dropped entirely, since the description implied by the term is inaccurate. However, the comparative anatomic significance of the term, properly used, should be considered first.

In actual gonioscopic examination one begins at the iris and moves forward toward its insertion on the inner surface of the ciliary body and then on to the trabecula and the cornea. There is first seen (fig. 2) an abrupt transition from the last roll of the iris to the lateral wall of the angle, usually obscuring the bow-shaped angle recess. Immediately above the transition line is the anteromedial surface of the ciliary body as it sweeps toward its insertions in the scleral spur and the corneoscleral trabecula. The gonioscopic appearance of the ciliary body and its inner covering of pectinate ligament tissue depends on the amount



A B

Fig. 2.—Histologic (*A*) and gonioscopic (*B*) appearance of the normal chamber angle in a blue-eyed patient. The section is from an eye which was treated with physostigmine salicylate.

of pigment in the uveal tissues of the eye. In blue-eyed patients, ordinarily neither the ciliary body nor the iris processes stand out as in brown eyes; so this area has a more or less diffuse light orange background on which white or gray iris processes may be seen (figs. 3 and 10 *A*). In brown-eyed patients the iris processes, when prominent, stand out as a dark brown forest against a slightly lighter brown background of ciliary body (fig. 10 *B*). The processes are always prominent in the eyes of Negroes but not always in those of less pigmented peoples. Sometimes they cannot be discerned in light brown or blue eyes. Among the processes in lightly pigmented eyes are sometimes seen delicate blood vessels.

There are only two important landmarks in the wall of the angle. The first is the anterior edge of the ciliary body. This corresponds to

the position of the scleral spur, which is sometimes seen as a thin white line just anterior to the ciliary body. The second landmark is the anterior edge of the trabecula, which corresponds histologically to the location of the circularly fibrillated anterior border ring of Schwalbe and the apparent termination of Descemet's membrane. In gonioscopic terminology this second landmark is usually called by its histologic designation, the anterior border ring of Schwalbe. It is glistening white and stands out in contrast to the transparent posterior layers of the cornea, which begin immediately in front of it. An illusion of protrusion of the Schwalbe line is produced by the fact that the line is the apex of an angle whose sides are formed by the inner surface of the trabecula and by the opaque limbus which runs from the end of Bowman's membrane to the end of Descemet's membrane.

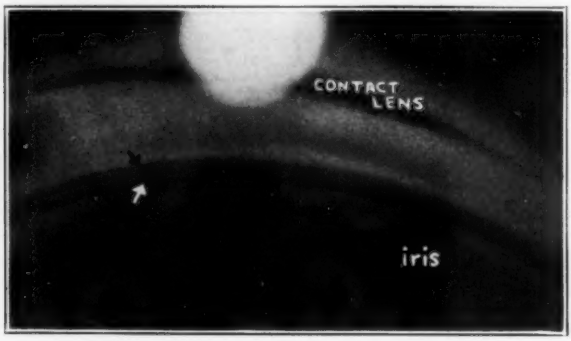


Fig. 3.—Lightly pigmented eye. The angle shows little differentiation between the ciliary body and the trabecula. The arrows point out the width of the angle wall. A small amount of pigment is visible on the trabecula. This and all other black and white photographs of the chamber angle are copies of kodachrome transparencies. The large white spot is the light reflex. The contact lens itself causes the dark zone in the uppermost portion of all the photographs of the angle.

Between the two landmarks lies the light tan corneoscleral trabecula, a band about 0.6 to 0.7 mm.[1] in width. Schlemm's canal lies deep to the posterior portion of the trabecula. Occasionally, probably as a result of pressure by the contact lens, blood can be seen in the canal in a normal eye. In spite of its presence in the canal, the blood has never been seen to enter the chamber itself. This finding seems to lend support to Friedenwald's [9] contention that there are no direct communications between the trabecular spaces and Schlemm's canal. In some eyes, usually brown, extensions of the iris processes onto the trabecula are seen. They may even reach the beginning of the cornea.

9. Friedenwald, J. S.: Circulation of the Aqueous: V. Mechanism of Schlemm's Canal, Arch. Ophth. **16**:65 (July) 1936.

Sometimes only isolated islands of iris processes may be seen on the trabecula, unconnected with the portion covering the angle recess and the ciliary body.

Just in front of the ring of Schwalbe one sees a band of meridional blood vessel loops, which are branches of the rami recurrentes anteriores of the anterior ciliary vessels. Since the sclera overlaps the transparent cornea by 1 to 2 mm., the vessel loops can be seen gonioscopically through the transparent posterior corneal layers but cannot be seen in ordinary external examination. These vessels are the ones which proliferate in cases of deep corneal vascularization (fig. 10 *C*).

Most anteriorly one sees the inner concave dome of the cornea ending in its yellowish optical section.

In myopic eyes the iris is inserted relatively far back on the ciliary body, so that much more of the band of the ciliary body is visible gonioscopically and the anterior chamber is therefore relatively deep. In hyperopic eyes the opposite is true; i. e., there is relatively less visible ciliary body and the anterior chamber is consequently shallower. In many such eyes the ciliary body is not visible though the angle is open. This obscuration of the ciliary body in eyes with shallow angles leads the inexperienced to conclude that peripheral anterior synechiae are present. By careful examination with a binocular microscope and with partial transillumination of the angle, these eyes are seen to be free of adhesions involving the trabecula, at least. These variations in the position of the iris insertion are probably the anatomic basis for the relative frequency of acute glaucoma in highly hyperopic persons and its relative infrequency in myopes.

In some middle-aged and senile persons deposits of pigment in the area of the corneoscleral trabecula covering Schlemm's canal may give rise to the appearance of a brown ring. Finer pigment deposits are often present also on the anterior ring of Schwalbe, particularly in the lower portion of the angle, due undoubtedly to gravitational factors. These pigment deposits are derived from the pigment epithelial cells on the posterior surface of the iris.

Because of the fact that the angle is seen from a considerably oblique position, especially in eyes with shallow angles, the trabecula is seen to be considerably foreshortened. Similarly, when pigment is observed on the trabecula over Schlemm's canal or when blood is seen in Schlemm's canal in an eye with a shallow angle, the pigment or blood may appear to lie in the anterior portion of the trabecula because of the oblique angle of observation.

It is obvious that miotics and mydriatics have a considerable effect on the visibility of the landmarks of the chamber angle. Dilatation of the pupil is associated with an increase in thickness of the iris tissues and thus causes a decrease in depth of the angle. Miosis, conversely,

is associated with stretching of the iris tissues and a comparative increase in depth of the angle. Gonioscopic observations are best made in the presence of miosis.

Figure 1 is a photomicrograph of an eye which was under the influence of atropine sulfate at the time of enucleation. Figure 2 shows a section of an eye under the influence of physostigmine salicylate.

GONIOSCOPIC TECHNIC

Observation of the chamber angle requires the use of a gonioscopic contact lens, a magnifier for the observer, an illuminating device and a fluid to fill the space between the contact lens and the cornea.

Of the contact lenses used, the best (but the most expensive) are the two made of ophthalmic glass, the Koeppe "A" lens and the more convex "C" lens (fig. 4). The "A" lens has a central depression in its surface. This was originally used to help hold the lens in place. For

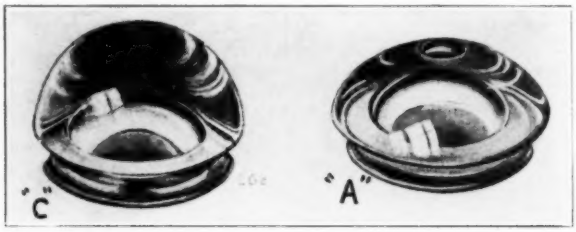


Fig. 4.—Contact lenses used in gonioscopic examination (Troncoso).

goniophotography or demonstration purposes the "C" lens, without any depression, is better. The optics of the thinner "A" lens are slightly better than those of the "C" lens; the former, therefore, is better to use in studying eyes with very shallow chambers. I have found this to be true by using both lenses on the same eyes. With the "C" lens the angle may appear to be obliterated, while with the "A" lens a thin slit of open angle may be discerned.

Another factor of importance in the choice of gonioscopic lenses is the diameter of their lips. The lips of the "C" lens and of the Zeiss "A" lens are too wide (22 mm.) for some palpebral fissures. The "A" lens made by Bausch and Lomb is smaller (20.8 mm.) and is often useful when the others cannot be inserted.

Barkan [10] has introduced a new type "A" lens made of plastic material. It is inexpensive and unbreakable and has an excellent stem attachment (fig. 5). It does not "wet" as well as does glass and scratches more easily. Its lips are as large as those of the Zeiss "A" lens.

10. Barkan, O.: A Diagnostic Contact Lens Made of Lucite, Arch. Ophth. **24**: 798 (Oct.) 1940.

For magnification one may use an ordinary binocular loupe or a corneal microscope. The loupe is satisfactory for determination of the results of operation and for examining aphakic eyes. For accurate observations as to the presence or absence of synechiae, however, one must use a microscope. The binocular corneal microscope is most satisfactory, but a monocular instrument, such as that recently devised by Troncoso, is less expensive and lighter in weight. It is to be recommended, especially for those who do not already have a corneal microscope available. The original gonioscope of Troncoso [5] is no longer manufactured.

The microscope may be held in the braced hands or may (preferably) be suspended from or carried on a gonioscopic support, such as that of Barkan,[11] that of Fine,[12] that of Hartshorne,[13] that of McAlester,[14] that

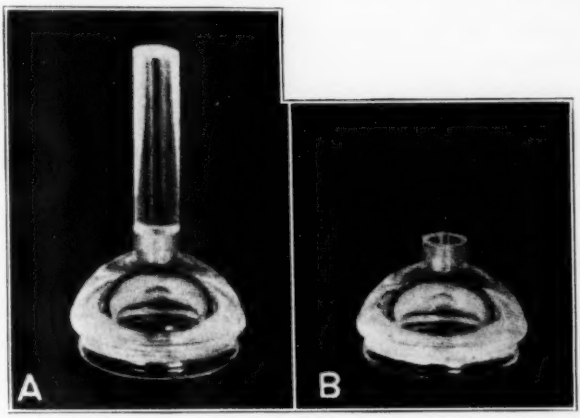


Fig. 5.—Barkan's plastic contact lens. *A*, with the handle attached. *B*, without the handle. (From Barkan.[10])

of McLean[15] or that of Bogart.[16] Gradle's balanced wall bracket support is convenient.

11. Barkan, O.: Recent Advances in the Surgery of Chronic Glaucoma, Am. J. Ophth. **20**:1237, 1937.

12. Fine, M.: Simple Illuminating Device for Gonioscopy with Binocular Microscope, Am. J. Ophth. **20**:943, 1937.

13. Hartshorne, I.: Simplified Portable Gonioscopic Unit, Am. J. Ophth. **21**: 545, 1938.

14. McAlester, A. W., III: Gonioscopy as an Aid to Localizing Small Foreign Bodies in the Anterior Chamber, Am. J. Ophth. **21**:1380, 1938.

15. McLean, J. M., and Goebel, A.: New Model Gonioscope, Arch. Ophth. **19**:983 (June) 1938.

16. Bogart, D.: A New Binocular Gonioscopic Apparatus, Arch. Ophth. **21**:709 (April) 1939.

The Barkan lamp, any other similar focal lamp or a coaxial lamp of the type used by Castroviejo may be used to illuminate the angle. It should preferably be attached directly to the microscope.

One may use water or physiologic solution of sodium chloride to fill the space between the cornea and the contact lens, but I have found the Gifford sodium carbonate buffer solution,[17] with a p_H of 7.3, most satisfactory. Less epithelial edema attends its prolonged use.

Observations of the chamber angle are usually made with the patient recumbent. After anesthesia has been induced with a drop or two of 0.5 per cent pontocaine hydrochloride solution, the lower lip of the contact lens is inserted under the edge of the lower lid. The patient is asked to look downward, and the upper lid is pulled over the upper lip of the lens. The patient's head is then turned to the opposite side, and the temporal edge of the lens is lifted slightly. The buffer solution is introduced with a dropper until no air bubbles remain under the lens. Slight pressure is made on the lens momentarily, and the patient's head returned to its original position.

A magnification of 10 to 14 diameters is best for ordinary gonioscopic technic. The contact lens itself magnifies the region of the angle approximately 1.3 times.

For slit lamp observation of the angle the flat-surfaced plastic lens of Goldmann[18] is satisfactory.

GONIOSCOPIC APPEARANCE OF THE CHAMBER ANGLE IN CASES OF PRIMARY GLAUCOMA NOT TREATED BY OPERATION

Peripheral anterior synechiae are the only distinguishing gonioscopic sign of primary glaucoma. When they are present, they are recognized by obliteration of one or both landmarks of the angle wall, namely, the anterior edge of the ciliary body and the line of Schwalbe. They may be either partial or total, and they may be ciliary, trabecular or corneal. They may be easily diagrammed if the landmarks are indicated by circles (fig. 6), as suggested by Kronfeld.

Knies[19] and Weber[20] independently discovered peripheral anterior synechiae in eyes which were enucleated after long-standing glaucoma and assumed that the synechiae were the cause of the glaucoma. Troncoso[21] refuted this idea and showed that the synechiae were really the result of the ocular hypertension. This has been substantiated by all subsequent investigators.

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17. Gifford, S. R.: Buffer Solutions, Arch. Ophth. **13**:78 (Jan.) 1935.
18. Goldmann, H.: Zur Technik der Spaltlampenmicroskopie, Ophthalmologica **96**:90, 1938.
19. Knies, M.: Ueber das Glaucom, Arch. f. Ophth. (pt. 3) **22**:163, 1876.
20. Weber, A.: Die Ursache des Glaucoms, Arch. f. Ophth. (pt. 1) **23**:1, 1877.
21. Troncoso, M. U.: Gonioscopy, Tr. Internat. Cong. Ophth. **1**:25, 1934.

Peripheral anterior synechiae are present commonly only in cases of congestive glaucoma and in cases of noncongestive glaucoma in the later stages. They are unquestionably the result of hyperemia and edema of the ciliary body in the former group and probably also in the latter. Friedenwald and Pierce [22] injected histamine into the vitreous of dogs and precipitated attacks of acute congestive glaucoma. The ocular findings in their experiments suggest the relation of experimental acute glaucoma to acute glaucoma in man.

Before the different types of primary glaucoma are discussed, a consideration of the depth of the anterior chamber and its relation to the depth of the angle is relevant. Rosengren,[23] using a highly accurate method of measurement, found that in the majority of a group of 104 glaucomatous eyes the depth of the chamber compared favorably with that observed in 810 normal eyes, except for the glaucomatous eyes which had very shallow chambers. Most of the latter were the eyes of

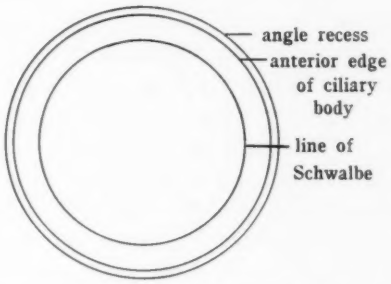

Fig. 6.—Schematic representation of the relations of the angle recess, the anterior edge of the ciliary body and the line of Schwalbe.

patients with congestive glaucoma. In a comparison of 100 normal eyes of patients over 55 years of age with 100 eyes of patients with noncongestive glaucoma and 15 with congestive glaucoma (with use of the Ulbrich drum) Gradle and I [24] made similar observations. We then compared the depth of the chamber in the glaucomatous eyes with the depth of the angle in the same eyes and concluded that, although there is a gross relation between the two, the relation is not absolute. The depth of the angle is the more important, since cases are observed in which the angle is extremely shallow and yet the chamber itself is relatively deep.

22. Friedenwald, J. S., and Pierce, H. F.: The Pathogenesis of Acute Glaucoma: II. Experimental Study, Arch. Ophth. **3**:574 (May) 1930.

23. Rosengren, B.: Studien über die Tiefe der vorderen Augenhammer, Acta ophth. **8**:99, 1930; **9**:103, 1931.

24. Gradle, H. S., and Sugar, H. S.: Concerning the Chamber Angle: III. A Clinical Method of Goniometry, Am. J. Ophth. **23**:1135, 1940.

The goniometric studies were made with a Koeppe contact lens and a micrometer graticule etched in tenths of a millimeter in one of the × 5.5 binocular microscope oculars. With the angle of observation as nearly parallel to the plane of the iris as possible, the length of an imaginary line between the line of Schwalbe and the point on the iris perpendicularly opposite was measured directly. The value obtained was then corrected by using the graticule-microscope magnification factor and the 1.3 magnification value for the contact glass. The values thus obtained were not considered as anatomic values because of the foreshortening of the obliquely seen space and the necessity of viewing the angle from somewhat above the plane of the iris; however, they were taken as values which would be useful in comparing the depth of the angle in different eyes and in the same eyes at various times.

1. *Congestive or Uncompensated Glaucoma.*—Salzmann,[3] the first to use the gonioscope in the study of glaucoma, found the chamber angles partly closed in 3 eyes with congestive glaucoma. Later, Troncosco,[25] in studies of acute or subacute congestive glaucoma observed 7 cases (26 per cent) in which the angles were open, 12 (46 per cent) in which they were partly closed and 7 (26 per cent) in which they were entirely closed. In studying chronic congestive glaucoma he observed 2 cases (25 per cent) in which the angles were open, 2 (25 per cent) in which they were partially closed and 4 (50 per cent) in which they were totally closed. He concluded that in the beginning of acute glaucoma the angle is open and that it closes later if the attack is severe or persistent. Thorburn[26] found synechiae in 3 of 6 cases of congestive glaucoma. Werner[27] found the angle obliterated by adhesions in 1 acutely glaucomatous eye and partially closed in 2 eyes with chronic congestive glaucoma. McLean,[28] in 21 cases of chronic congestive glaucoma, found no synechiae in 4, minor synechiae in 6 and extensive synechiae in 11.

Of my series of 68 eyes with acute glaucoma, all with shallow chambers, none were examined during an acute attack. The tension was normalized within one to three days after the onset of the attack in 13 eyes (table 1). In 9 of the 13 eyes the angle was deep enough to be seen gonioscopically; 6 of these had entirely open angles, and 3 had angles partly open. The open angles were all in eyes in which the attack had been relieved within twenty-four hours of its onset. In the 3 with partly open angles the attack had lasted forty-eight to seventy-two hours.

25. Troncoso, M. U.: Closure of the Chamber Angle in Glaucoma, Arch. Ophth. **14**:557 (Oct.) 1935.

26. Thorburn, A.: A Gonioscopic Study of Anterior Peripheral Synechiae in Primary Glaucoma, Thesis, Stockholm, 1927.

27. Werner, S.: Gonioskopische Untersuchungen bei Glaucoma primarium, Acta ophth. **10**:427, 1932.

28. McLean, J. M.: Tr. Am. Acad. Ophth., 1940, to be published.

One of these had an open area involving about 25 degrees of the circumference of the angle. In this eye the tension was reduced to normal by miotics but subsequently rose again. The border of the pupil at the time of the low tension was flattened in the area corresponding to the opening of the angle. A chart of the tension (fig. 7) is shown. For all the remaining 55 eyes, miotics were used for two days to seven months without relief of the glaucoma. Surgical operations were performed on 41 eyes.

All evidence indicates that in cases of acute congestive glaucoma the angle is normal before the onset of the attack but is closed during the attack. If miotics are used early, before the adhesions are firm, at least a portion of the angle is opened and the tension is reduced to normal. If

TABLE 1.—*Acute Glaucoma Relieved by Miotics*

Patient	Angle Before Attack	Duration of Attack	Angle After Normalization with Miotics
1. M. C.	Open	1 day	Open
2. B. D.	.....	3 days	Open; trabecular adhesions in places
3. T. R.	.....	2 days	Too shallow
4. L. S.	Open	1 day	Open
5. A. T.	.....	1 day	Open
6. R. H.	.....	2 days	Too shallow
7. J. K. (RE)	.....	1 day	Open
8. J. K. (LE)	.....	1 day	Open
9. A. B.	.....	1 day	Too shallow
10. E. B.	.....	2 days	Open ⅗; too shallow ⅓
11. A. P.	.....	2 days	Small upper temporal area open; later tension rose
12. M. K.	.....	1 day	Open
13. A. K.	.....	3 days	Open 12 to 3 o'clock; closed elsewhere

the attack has persisted for over two or three days, the adhesions usually have become firm and miotics are of little or no avail. Operation must then be resorted to. Of my series of acutely glaucomatous eyes, iridectomy was done in 20, with success in 14 (70 per cent). Trephining operations were performed in 15, with success in 9 (60 per cent).

Comment on the surgical aspects of acute glaucoma will be reserved for the discussions of the individual operations.

Of 33 eyes with chronic congestive glaucoma, I found the angle too shallow for observation in 13, shallow but open in 19 and entirely closed in 1. In this group the same process in a milder degree than in the acutely glaucomatous eyes must be assumed to go on. Synechiae form if the congestive phase lasts long enough or is repeated sufficiently often.

When firm synechiae form in cases of congestive glaucoma, they may occasionally be detached with the use of miotics. In only 2 cases of the present series did this occur. In each of them portions of the anterior border layer of the iris remained attached to the trabecular wall.

2. *Noncongestive, Compe sated or Chronic Simple Glaucoma.*—In cases of early or moderately advanced noncongestive glaucoma, the angle is usually open and cannot be distinguished gonioscopically from the normal. In cases of late noncongestive or absolute glaucoma, however, adhesions form when for some reason or other the vascular or neurovascular controlling mechanism no longer functions adequately, and mild congestive episodes occur. Sometimes the onset of vascular decompensation is severe and is ushered in with symptoms and signs similar

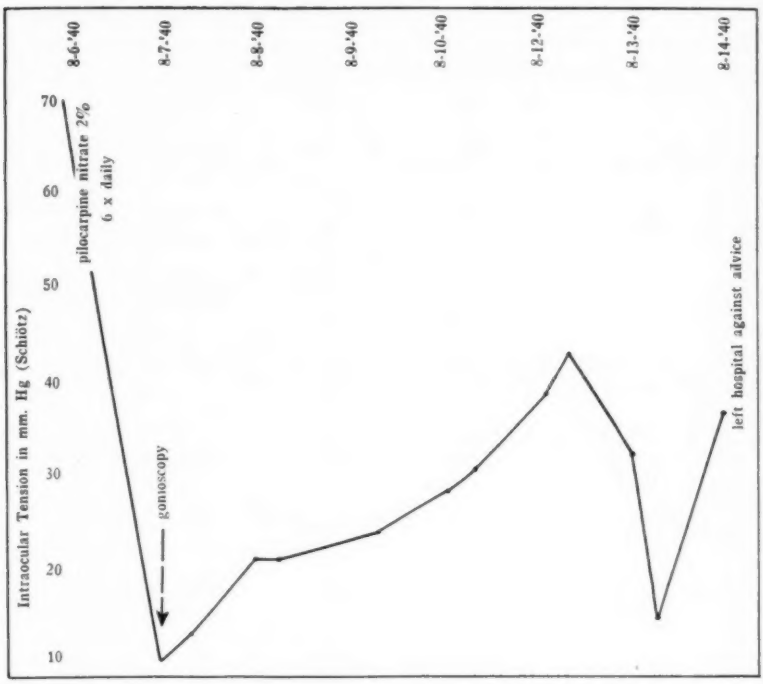

Fig. 7.—Tension curve in the case of patient A. P. A small area of open angle was present on gonioscopic examination.

to those of an acute congestive attack. In such circumstances the history is extremely helpful in the differential diagnosis.

Salzmann [3] examined 1 eye with simple glaucoma and found the angle entirely open. Troncoso,[25] in observing 34 eyes with noncongestive glaucoma, found 19 (56 per cent) with open angles, 13 (38 per cent) with angles partially closed and 2 (5.8 per cent) with angles totally closed. Thorburn,[26] in 9 cases of simple glaucoma, found 6 open angles, 1 angle partially closed with corneal adhesions and 2 angles partially closed with trabecular adhesions. Werner [27] found that 20 of 28 eyes had open angles; the remainder showed synechiae, 2 trabecular and 6

corneal. Barkan [7] stated that he found the angle open in 60 per cent of cases of this type of glaucoma. McLean,[28] in 66 cases of noncongestive glaucoma, found no synechiae in 74.3 per cent, minor synechiae in 22.7 per cent and extensive synechiae in 3 per cent.

In my series of cases of chronic noncongestive glaucoma (344 eyes) I found the angle open in 337 eyes (97 per cent). This series may be subdivided into four groups, according to the changes in the visual field: first, cases of early involvement, in which the visual fields showed minimal change; second, cases of moderately advanced disease, in which the fields varied in constriction up to 10 degrees from the fixation point; third, cases of late involvement, in which the fields were within the 10 degree circle, and fourth, cases of absolute glaucoma. In the first two groups (314 eyes) there were no peripheral anterior synechiae. In the third group (10 eyes) 3 eyes had open angles; 4 had partly open angles, and 3 had obliterated angles. In the cases of absolute glaucoma 20 eyes were examined. The angles were open in 7, partly open in 6 and obliterated in 7.

It must be concluded that in cases of noncongestive glaucoma, peripheral anterior synechiae occur only with late and absolute involvement. Further evidence of the fact that a congestive phase is necessary to bring about the formation of synechiae is the fact that in no case of simple glaucoma not operated on, without any further phase of decompensation, have I seen adhesions increase during the past two years.

Barkan [7] has attributed considerable etiologic significance to trabecular sclerosis and pigment granule blockage of the trabecular spaces. Sclerosis has not been recognized gonioscopically by subsequent writers (Sugar; McLean). I found pigment granules in 50 per cent of 199 eyes with noncongestive glaucoma.[1] Since pigment is also present in normal nonglaucomatous eyes of middle-aged and senile persons, though less frequently, it must be considered ordinarily of little significance except in the relatively few cases in which dense trabecular pigment deposits are present. It is conceivable that in these cases the trabecular spaces may be so filled with pigment that mechanical access of the aqueous to the osmotic influence of the fluid in Schlemm's canal is hindered.

GONIOSCOPIC APPEARANCE OF THE CHAMBER ANGLE IN CASES OF SECONDARY GLAUCOMA

Of 180 eyes with secondary glaucoma which were studied gonioscopically, the glaucoma in 70 was associated with capsular exfoliation; in 53 it followed removal of cataract; in 5 it was post-traumatic; in 8 it followed healed acute iridocyclitis; in 7 it followed occlusion of the central retinal vein; in 3 it was associated with diabetic rubeosis; in 16 it was associated with dislocated or subluxated lenses; in 3 it was asso-

ciated with intumescent lenses, and in 16 it was probably secondary to anomalies of the chamber angle.

1. *Glaucoma Capsulare.*—This type of glaucoma, often unrecognized and usually erroneously considered chronic simple glaucoma, occurs in eyes with exfoliation of the zonular lamella of the lens capsule. Exfoliated particles have been recognized on the trabecula histologically by Bey [29] and gonioscopically by Barkan [7] and by Gradle and me.[30] Large particles sometimes stand out as they lie on the line of Schwalbe or the trabecula. Finer particles of zonular lamella mixed with fine pigment granules give the ciliary body and the trabecula a faintly veiled appearance in lightly pigmented eyes. A characteristic trabecular pigment ring in the zone overlying Schlemm's canal is always present (fig. 8). This ring is due to deposition in the trabecular spaces of pigment which

Fig. 8.—Deposit of pigment on the trabecula in a case of capsular exfoliation. The area below the pigment line is the ciliary body. The arrows point to the pigment line.

has been rubbed off the pigment epithelium of the iris. In 11 cases of unilateral capsular exfoliation, the uninvolved eye in 9 showed no pigment ring, while the eye with exfoliation showed such a ring in every instance.

In cases in which iridectomy has been performed, involvement of the equatorial zonular lamella and the zonule fibers and deposits of exfoliated material on the ciliary processes can be seen. The equatorial exfoliation was reported by Gradle and me [30] and shortly afterward,

29. Bey, S.: A Contribution to the Study of Exfoliation of the Lens Capsule, Brit. J. Ophth. **16**:65, 1932.

30. Gradle, H. S., and Sugar, H. S.: Concerning the Chamber Angle: II. Exfoliation of the Zonular Lamella and Glaucoma Capsulare, Am. J. Ophth. **23**: 982, 1940.

independently, by Irvine.[31] Opacity of the zonule fibers was noted at the time of the first report of equatorial exfoliation. The presence of exfoliated material on the fibers has been known since its first description by Trantas.[32]

Irvine stated the belief that the exfoliated material is from the fibers themselves and not merely a deposit thereon, as was originally believed.

Adhesions are not usually present in the chamber angle in cases of glaucoma capsulare not treated by operation except in the few instances in which congestion has supervened in the presence of long-standing absolute glaucoma capsulare. Even after operations have been performed there is comparatively little tendency for the formation of peripheral anterior synechiae. In contrast to the findings (to be discussed) regarding the changes in the chamber angle following trephining operations, none of the eyes with glaucoma capsulare operated on showed complete obliteration of the angle. This absence of any considerable tendency to synechia formation is probably due to the insulating effect of the minute capsular particles on the angle wall.

One of the remarkable and heretofore neglected facts about glaucoma capsulare is that removal of cataractous lenses usually has no great effect on the ocular hypertension. Of 12 eyes from which the lenses were removed, all had ocular hypertension (pressure over 30 mm. of mercury [Schiötz]), at least once postoperatively. Fortunately the tension in 10 was controlled with miotics; only 2 required cyclodialysis. The angles were open, below at least, with the occasional exception of very fine ciliary synechiae, in all cases (table 2).

2. *Glaucoma Following Surgical Treatment of Cataract.*—Secondary glaucoma following removal of cataract has been known ever since the first report of Bowman (1865) to every operator who has observed a sufficient number of cases postoperatively. Czermak[33] concluded that this type of glaucoma is due to delayed closure of the wound, entailing prolonged absence of the anterior chamber. Histologically he found the iris adherent to the periphery of the cornea, the original chamber angle having become obliterated.

In a previous paper[1] I pointed out the gonioscopic aspects of this type of glaucoma. In a series of 30 cases, the angle "in 29 was completely obliterated. In one, a small portion of the angle was open and the tension became normal with miotics. . . . Many of the histories indicated a prolonged postoperative period during which the anterior chamber was collapsed, associated in some instances with recognized

31. Irvine, R.: Tr. Pan-Am. Cong. Ophth., 1940, to be published.

32. Trantas, M.: Lésions séniles de la capsule antérieure du cristallin, Arch. d'opht. **46**:482, 1929.

33. Czermak, W.: Die augenärztliche Operationen, Vienna, C. Gerolds Sohn, 1898.

choroidal detachment or iris prolapse. Complete obliteration of the angle is probably the cause of this type of glaucoma." Later, Kronfeld and Grossmann [34] undertook to define more closely the relation between the three factors of delayed restoration of the anterior chamber, formation of peripheral synechiae and the occurrence of glaucoma. As part of their work they examined 13 persons with aphakia in whom delay in formation of the anterior chamber after operation for cataract had been noted. These patients were selected from a series of 120 unselected surgical patients with aphakia. In 2 patients with extensive synechiae glaucoma developed while they were under observation. Two

TABLE 2.—*Results of Removal of the Lens in Cases of Glaucoma Capsulare*

Patient	Highest Preoperative Tension, Mm. Hg (Schiötz)	Type of Operation	Description of Chamber Angle After Operation	Highest Postoperative Tension, Mm. Hg	Means Used to Relieve Tension
1. C. G.	45	Extracapsular	(1937)	52	Cyclodialysis plus miotics
2. J. F.	60	Extracapsular	Partly open	38	Miotics
3. G. D. (R)..	32	Intracapsular (vitreous loss)	Partly open; obliterated only in wound area	37.5	Miotics
4. G. D. (L)..	76	Intracapsular (vitreous loss)	Palpebral fissure too small	46	Cyclodialysis repeated
5. B. P. (R)..	26	Intracapsular	Partly open	32	Miotics
6. B. P. (L)..	32.5	Loop	Open below; obliterated in upper half	32.5	Miotics
7. F. U. (R)..	45	Extracapsular	Partly open	33	Miotics
8. F. U. (L)..	32	Extracapsular	Partly open	35	Miotics
9. J. P.	48	Extracapsular	Open below; closed in upper half	53	Miotics
10. M. S.	19	Extracapsular	Partly open, partly trabecular adhesions	52	Miotics
11. T. F. (R)..	35	Intracapsular	Not seen	30.5	Miotics
12. T. F. (L)..	48	Extracapsular	Partly open	30.5	Miotics

other patients with borderline conditions were noted. The authors also studied 32 cases of this type of glaucoma (partly from the same material previously used in my report of 30 cases) and concluded that "delayed closure of an operative incision actually leads to peripheral anterior synechiae of an extent depending upon the amount of the delay, and perhaps upon the texture of the anterior border layer of the iris. The occurrence of glaucoma in such cases is shown to depend upon and be secondary to the presence of extensive peripheral anterior synechiae."

In the present study, 52 eyes with secondary glaucoma following operation for cataract were studied. Cases in which uveitis was present and cases in which the interval between operation for cataract and

34. Kronfeld, P. C., and Grossmann, E. E.: The Relation of the Gonioscopic Findings to the Incidence of Secondary Glaucoma in Operative Aphakes, Tr. Am. Acad. Ophth., 1940, to be published.

development of ocular hypertension was over a year were excluded, since in some cases of this type the glaucoma may really be primary.

The 52 eyes may be divided into two groups: In the first (12 eyes) the glaucoma was first noted after discission of an after-cataract (table 3), and in the second (40 eyes) the glaucoma followed the removal of the lens (table 4). In each case the tonometric tension was normal prior to operation.

TABLE 3.—*Secondary Glaucoma Following Discission of After-Cataract*

Patient	Type of Cataract Operation	Interval Between Operation and Formation of Anterior Chamber	Interval Between Cataract Operation and Discission	Interval Between Discission and First Evidence of Glaucoma	Chamber Angle	Means Used to Relieve Glaucoma
1. T. L.	Extracapsular	3 weeks	11 weeks	5 months	Obliterated	Operation
2. C. C.	Extracapsular	1 day (hyphema 6th day)	7 weeks	8 weeks	Nearly obliterated; anterior part of trabecula visible	Operation
3. M. K.	Extracapsular	5 days	6 weeks	2 days	Trabecular adhesions 2 to 8 o'clock; obliterated elsewhere	Miotics
4. J. B.	Loop; extracapsular	2 days	3 months	2 months	Obliterated	Operation
5. L. C.	Extracapsular	?	5 weeks	6 weeks	.....	Operation
6. A. F.	Extracapsular	4 days	10 weeks	10 weeks	Obliterated	Miotics (poor physical condition)
7. A. S.	Extracapsular	?	3 months	6 weeks	Trabecular adhesions 3 and 8 o'clock; obliterated elsewhere	Miotics
8. E. J.	Extracapsular	?	10 weeks	5 months	Trabecular adhesions 6 to 8 and 2 to 4 o'clock; obliterated elsewhere	Operation
9. W. M.	Extracapsular	3 days	5 weeks	19 months	Obliterated	Operation
10. A. P.	Extracapsular	10 days	5 months	8 months	.....	Operation
11. A. S.	Extracapsular	?	2 months	2 weeks	Obliterated	Operation
12. C. L.	Extracapsular	6 days	7 weeks	11 months	Trabecular adhesions below; obliterated above	Miotics

In the second group all but 2 eyes had peripheral anterior synechiae extending at least up to the anterior third of the trabecula and usually up to the line of Schwalbe. In the 2 exceptions only small areas remained open. The tension in both was normalized with miotics.

In cases of this type all the blame for the formation of synechiae has been hitherto placed on postoperative collapse of the anterior chamber. The overuse of atropine sulfate after operation and the organization of repeated hemorrhages may be additional factors in their formation.

When one finds a fairly large area of open angle in an eye with secondary glaucoma following removal of the lens, it is probable that

TABLE 4.—*Noninflammatory Secondary Glaucoma Following Operations for Cataract*

Patient	Type of Operation	Interval Before Formation of Anterior Chamber	Interval Between Cataract Operation and First Evidence of Glaucoma	Chamber Angle	Means Used to Relieve Glaucoma
1. P. N.....	Intracapsular	5 days; collapsed on 10th day; choroid detached	1 month	Obliterated; ointment globule in anterior chamber	Part of oil removed through keratome incision; operation
2. J. M.....	Extracapsular	4 flaps over iris prolapse	2 months	Grossly obliterated	Operation
3. S. L.....	Intracapsular	3 flaps over iris prolapse	2 months	Grossly obliterated	Uncontrolled
4. E. B	Extracapsular	Hyphemia 6 weeks	6 weeks	Obliterated	Uncontrolled
5. J. C.....	Extracapsular	1 day; cortical remnants	7 months	Obliterated	Operation
6. J. B.....	Extracapsular	Prolapse	2 months	Obliterated	Uncontrolled
7. T. L.....	Intracapsular	7 days; prolapse; anterior chamber collapsed on 10th day on removal of suture	9 weeks	Trabecular adhesions below; closed elsewhere	Operation
8. M. H.....	Extracapsular	5 days; prolapse	Occasional tension 28; once 37	Obliterated	Filtering cicatrix ?
9. A. K.....	Extracapsular	3 days	5 weeks	At least ⅔ trabecula covered by iris all around	Operation
10. H. G.....	Extracapsular	13 days	5 weeks	Obliterated	Operation
11. M. M.....	Extracapsular	Trauma; hyphemia 7th day	6 weeks	Obliterated	Operation
12. M. M. (R)	Extracapsular	6 days	3½ months	Obliterated	Miotics
13. M. M. (L)	Extracapsular	12 days	6 weeks	Obliterated	Operation
14. L. R. (R)	Intracapsular	4 days	1 month	Obliterated	Filtering cicatrix ?
15. L. R. (L)	Intracapsular	4 days; collapsed on 6th day; reformed 11th day; choroid detached	6 weeks	Small opening 7 to 8 o'clock; obliterated elsewhere	Filtering cicatrix ?
16. D. S.....	Intracapsular	14 days; choroid detached	3 months	Obliterated except at 5:30 o'clock	Miotics
17. M. M.....	Extracapsular	5 days; choroid detached in 8 days	10 weeks	Obliterated	Operation
18. A. S.....	Extracapsular	19 days	7 months	Obliterated	Operation
19. H. S	Intracapsular	8 days	3 months	At least ⅔ trabecula below obliterated; obliterated above	Operation
20. C. S.....	Extracapsular	4 days; hyphemia 5th day	3 months	Trabecular adhesions in lower temporal area; obliterated elsewhere	Filtering cicatrix ?
21. J. S.....	Intracapsular	5 days	5 weeks	Obliterated	Filtering bleb
22. T. D.....	Extracapsular	1 day	1 month	Open 12:30 and 6 o'clock; obliterated elsewhere	Miotics
23. J. G.....	Extracapsular	4 days	1 month	Obliterated	Operation
24. L. H.....	Extracapsular	7 days; prolapse flap 5th week	1 month	Obliterated	Operation (loosening of prolapse)

TABLE 4.—*Noninflammatory Secondary Glaucoma Following Operations for Cataract—Continued*

Patient	Type of Operation	Interval Before Formation of Anterior Chamber	Interval Between Cataract Operation and First Evidence of Glaucoma	Chamber Angle	Means Used to Relieve Glaucoma
25. M. K.....	Extracapsular	3 days	6 months	Trabecular adhesions 5 to 7 o'clock; obliterated elsewhere	Uncontrolled
26. S. M.....	Extracapsular	?	2 months	Obliterated	Operation
27. R. B.....	Extracapsular	7 days	3 weeks	Open 11 to 12 o'clock.	Normal with eserine (usually)
28. B. C. (R)	Extracapsular	3 days	4 months	Obliterated	Operation
29. B. C. (L)	Loop	?	1 month	Obliterated	Operation
30. A. M.....	Loop, intracapsular	?	7 months	Obliterated	Operation
31. J. L.....	Extracapsular	2 days	1 month	Obliterated	Operation
32. E. B.....	Extracapsular	Iris incarcerated	7 weeks	Obliterated	Uncontrolled
33. C. C.....	Extracapsular	7 days	5 months	Obliterated	Operation
34. H. D.....	Extracapsular	12 days	3 months	Obliterated	Filtering cicatrix ?; miotics
35. N. E.....	Intracapsular	7 days; hyphemia	7 months	Obliterated	Operation
36. H. F.....	Extracapsular	10 days	1 month	Obliterated	Operation
37. L. H.....	Extracapsular	10 days	1 month	Practically obliterated	Operation
38. J. W.....	Extracapsular	13 days	1 month	Obliterated	Operation
39. V. D.....	Extracapsular	12 days	3 months	Obliterated	Tension 37.5 once; filtering cicatrix ?
40. E. R.....	Extracapsular	12 days	6 weeks	Obliterated	Operation

TABLE 5.—*Glaucoma Following or Associated with Uveitis in Patients with Postoperative Aphakia*

Patient	Type of Cataract Operation	Interval Before Formation of Anterior Chamber	Interval Between Cataract Operation and First Evidence of Glaucoma	Complications	Description of Chamber Angle	Means Used to Relieve Glaucoma
1. H. M.	Extracapsular	3 days	2 months	Iridocyclitis	Open; adhesions at 5 and 6 o'clock	Treatment of uveitis
2. M. P.	Intracapsular	?	5 months	Marked iridocyclitis	Open below	Cyclodialysis
3. E. C.	Extracapsular	?	10 years	Chronic cyclitis with marked mutton-fat deposits	Open 2 to 3:30 o'clock; obliterated elsewhere	Miotics
4. F. C.	Extracapsular	3 days	1 month	Had cortex in anterior chamber	Partly open	Cyclodiathermy operation
5. E. J.	Intracapsular	2 days	5 months	Healed iridocyclitis	Partly open	Miotics

the cause falls into one of the four following groups: first, cases of glaucoma capsulare, as discussed in the previous section; second, cases of active or healed uveitis; third, cases of probable primary glaucoma in which the operation for cataract was merely incidental, and fourth, cases of epithelial ingrowth.

Table 5 illustrates, for comparison, the data concerning the chamber angle in a group of glaucomatous eyes with active or healed uveitis following operation for cataract. In all but 1 of them a large part of the angle was open.

The occurrence of several cases of surgical aphakia with completely closed chamber angles and no signs of glaucoma or only occasional rises in tension was disturbing until it was found that in each instance incarcerated iris tissue was present in the wound and that a filtering bleb or cicatrix was the probable cause of the absence of glaucoma. Cases 8, 14, 15, 20, 34 and 39 were such cases. In case 21 a definite filtering bleb was present. In 1 other case of postoperative aphakia with the angle closed there was never any tension over 28 mm. of mercury (Schiötz), although the patient was examined daily for two months after a conjunctival flap was drawn over the area of the incision. In this case the chamber had formed on the second postoperative day and was flat on the fourth day. It remained flat for twenty-six days and reformed only after the flap was made. A choroidal separation was visible while the chamber was collapsed. A fistula, undoubtedly very minute, probably caused the original collapse of the chamber and the persistent normal tension.

Discission as a cause of glaucoma following removal of senile cataract has never been studied gonioscopically. In my series of 12 cases there was no evidence of increased tension before the discission was performed. Since there is already evidence that anything which causes collapse of the anterior chamber, especially when any irritation of the ciliary body is added, is apt to result in formation of or increase in peripheral anterior synechiae, one must consider that this operation may be the precipitating factor in this form of glaucoma, especially if the injudicious postoperative use of atropine is added. Similarly, discission may precipitate glaucoma in children on whom repeated needlings for congenital cataract are done.

The fact that closure of the chamber angle in itself gives rise to glaucoma is highly significant from the point of view of human physiology. It adds emphasis to the views of Leber,[35] Friedenwald and

35. Leber, T., in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1903, vol. 2, pt. 2.

Pierce,[36] and others, who have expressed the belief that Schlemm's canal is the chief structure concerned in reabsorption of water from the eye.

One means of facilitating the formation of the anterior chamber and thereby preventing the formation of synechiae is the use of a corneoscleral suture at the time of operation. In a paper on that subject by Leech and me,[37] the complications in three separate groups of 150 cataract operations each were compared. The first group was done without sutures, the second with conjunctival sutures and the third with corneoscleral sutures. Delayed formation of the anterior chamber occurred in 7.3 per cent of the first group, 6 per cent of the second and 2.6 per cent of the third. McLean,[38] in describing a new corneoscleral suture introduced at the Wilmer Institute, found delayed formation of the anterior chamber or delayed closure of the wound after 14.1 per cent of 64 operations for cataract done with conjunctival sutures and in 3.6 per cent of 110 operations done with the new suture. All the operations were performed by McLean himself, so that an excellent series is provided for comparison under uniform conditions.

Cyclodialysis has been accepted by most operators as the procedure of choice for treatment of glaucoma following removal of the lens. As a matter of fact, it is the only operation for glaucoma which can safely be done in an aphakic eye. The site of cyclodialysis for this type of glaucoma may be anywhere along the circumference of the angle. I prefer to avoid the site of the iridectomy, since the limbs of the iris are adherent or incarcerated there. Since one of the goals of good surgical technic is to obtain the desired result without undue trauma or hemorrhage, the areas of least adhesion are the areas of choice.

In my series of cases of secondary glaucoma following operation for cataract, 56 cyclodialyses were done. Twenty-three (41 per cent) were successful, and 33 were unsuccessful. Further consideration will be given to this operation in a later section.

3. *Post-Traumatic Glaucoma.*—In a small group of 5 cases of nonperforating trauma followed by glaucoma, the angle was found to be open in every case. The group includes only those cases in which evidence of the acute traumatic reaction was no longer present and in which no dislocation of the lens was demonstrable. No conclusions can be drawn except that there is no visible mechanical closure of the chamber angle in

36. Friedenwald, J. S., and Pierce, H. F.: Circulation of the Aqueous: II. Mechanism of Reabsorption of Fluid, Arch. Ophth. **8**:9 (July) 1932.

37. Leech, V. M., and Sugar, H. S.: Reduction of Postoperative Complications in Cataract Operations with Corneoscleral Sutures, Arch. Ophth. **21**:966 (June) 1939.

38. McLean, J. M.: A New Corneoscleral Suture, Arch. Ophth. **23**:554 (March) 1940.

cases of nonperforating trauma. Open angles were likewise found by Troncoso [21] in similar cases.

It is obvious that in cases of perforating trauma the angle will be involved to an extent depending on the duration of collapse of the chamber and the degree of exudative reaction.

4. *Healed Acute Iridocyclitis with Late Development of Glaucoma.*—In cases of this type, not including cases of iris bombée, the angle is usually partly or entirely open but covered with spots of pigment and cellular debris. When synechiae are found, they are circumscribed and appear to result from the organization of fibrinopurulent exudate (fig. 9). It is conceivable that organization of exudate within the trabecular spaces mechanically keeps the aqueous from access to the walls of Schlemm's canal.

Iris bombée is obviously associated with obliteration of the chamber angle and cannot usually be studied gonioscopically.

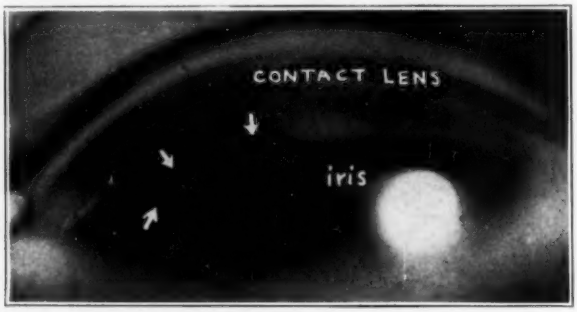

Fig. 9.—Localized dense synechia following organization of exudate in a patient with acute iridocyclitis.

Eyes with active acute or chronic iridocyclitis usually have open chamber angles.

5. *Hemorrhagic Glaucoma.*—(*a*) Hemorrhagic Glaucoma Following Occlusion of the Central Retinal Vein: In patients with branch obstruction or with partial obstruction of the central vein, no glaucoma developed in the involved eye and the angles were entirely normal in appearance. When secondary glaucoma develops in case of complete obstruction of the central vein, the angle remains open at first but appears to be very congested. A short time later the angle is found to be obliterated and many new anastomotic vessels are seen at the point where the iris becomes adherent to the cornea.

(*b*) Rubeosis Iridis: Kurz [39] described the gonioscopic appearance of the angle in this condition in 1939. In the 3 eyes observed by me the

39. Kurz, O.: Zur Rubeosis iridis diabetica, Arch. f. Augenh. **110**:284, 1937.

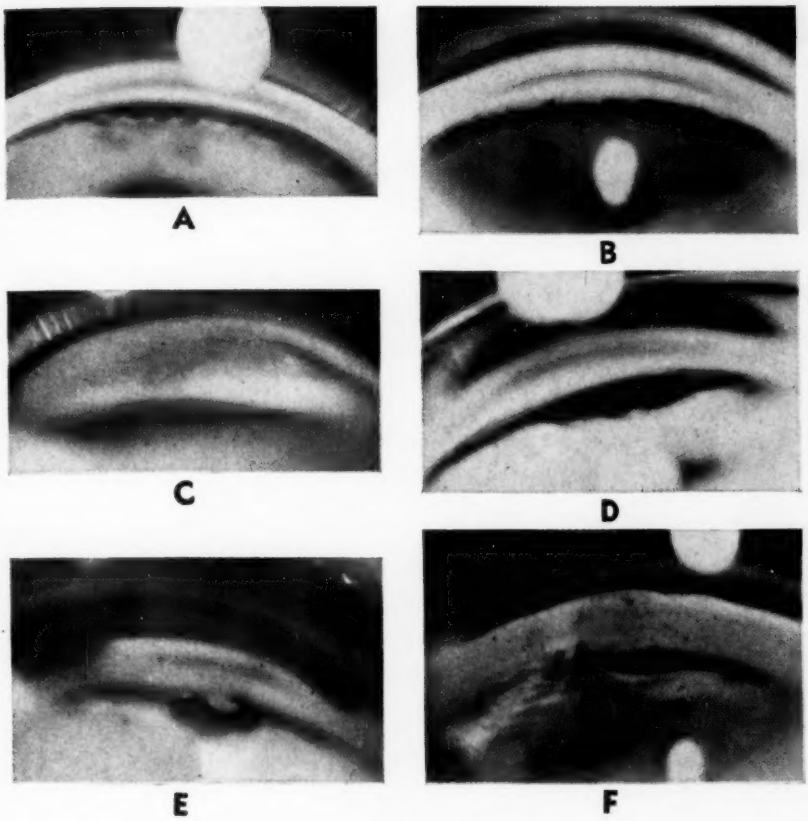

Fig. 10.—*A*, chamber angle in a lightly pigmented eye. Little differentiation is visible between the ciliary body and the trabecula, but a deposit of pigment is present over the area of Schlemm's canal. The area posterior to the pigment, between it and the last roll of the iris, is the ciliary body. The orange zone anterior to the pigment is the anterior portion of the trabecula. The contact glass is visible in the upper portion of the photograph. *B*, normal angle in a Negro patient. Many iris processes extending from the angle recess to the middle of the trabecula are seen. Differentiation of the ciliary body is difficult, since it is covered by the processes to a large extent. *C*, proliferation of the loops of the branches of the anterior ciliary vessels in a case of syphilitic interstitial keratitis. The trabecula is well shown. *D*, broad cyclodialysis cleft. *E*, open trephine hole. The scar of the peripheral iridectomy is visible. *F*, completely plugged trephine opening. The iris is seen to bulge forward into the opening.

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angles, like those described by Kurz, were completely obliterated. New anastomotic vessels covered the area of the new iris root in each case.

6. *Glaucoma, Associated with Dislocated Subluxated Lenses.*—Of 16 eyes examined, 13 showed wide open angles. In 2 subluxated lenses which apparently impinged on the back of the iris root below, a localized narrow area of adhesion was present. One other eye had fine trabecular adhesions all around the angle.

7. *Glaucoma Associated with Intumescent Lens.*—Acute glaucoma occurred in 3 eyes in which intumescence of the crystalline lens was present. In each of these simple mechanical closure of the chamber angle was found. Miotics were effective in relieving this type of glaucoma prior to operation for cataract. In 1 case the preoperative use of cocaine hydrochloride precipitated a second attack.

8. *Glaucoma Associated with Anomalies of the Chamber Angle.*—(*a*) Hydrophthalmia: The association of anomalies of the chamber angle with this form of glaucoma has been known since the observation of persistent mesodermal tissue in the angle by Horner in 1880 and of the absence of Schlemm's canal by Cross in 1896.

Earlier in this paper it was pointed out that in embryonic life the space which later corresponds to the chamber angle is filled by a mesodermal reticulum which undergoes partial atrophy and gives rise during the third to the fifth month to the anterior chamber and to the meshwork of the chamber angle. Any interference with the process of atrophy may give rise to anatomic and physiologic disturbances of the angle.

Anderson,[8] in his comprehensive monograph on this condition, attributed the glaucoma to one or more of the following causes:

1. Persistent or aberrant meshwork in the angle.
2. Poorly developed or absent canal of Schlemm.
3. Posteriorly placed (fetal) canal of Schlemm.
4. Rudimentary development of the scleral spur.
5. Peripheral anterior synechiae due to lack of differentiation or to postinflammatory synechiae.

In 4 cases of hydrophthalmia, gonioscopic study revealed the same picture in each case. The iris tissue, instead of inserting on the ciliary body, continued along the angle to the region of the line of Schwalbe, obscuring any evidence of the landmarks of the angle wall. Obliteration of the landmarks did not appear to be due to synechiae, since the iris markings continued over the entire tissue. Because of the relative transparency of this persistent tissue in 3 cases in which the irides were blue, the reflection of the pigment epithelium of the iris root onto the ciliary body could be seen. No conclusions as to the presence or absence of Schlemm's canal could be drawn from the gonioscopic appearance.

(*b*) Juvenile Glaucoma: In each of 5 cases of unilateral simple glaucoma in persons in their third decade of life, no anomalies of the chamber

angle could be seen. In another such case, that of an 11 year old white girl, the affected eye showed proptosis of 5.5 mm. The disk was excavated about 1.6 mm., and the chamber angle was entirely open and appeared normal. The intraocular tension during the preoperative period of observation varied constantly between 30 and 66 mm. of mercury (Schiötz), even after administration of physostigmine salicylate. It has been well controlled for the past three years by an Elliot trephining operation.

(*c*) Aniridia: The tendency for glaucoma to develop in eyes with aniridia in later life was recognized by Collins in 1891. Only 2 patients with this condition have been studied gonioscopically by me, 1 with glaucoma and 1 without. An iris stump was present all around the circumference of the angle in each case. In addition, persistence of meso-

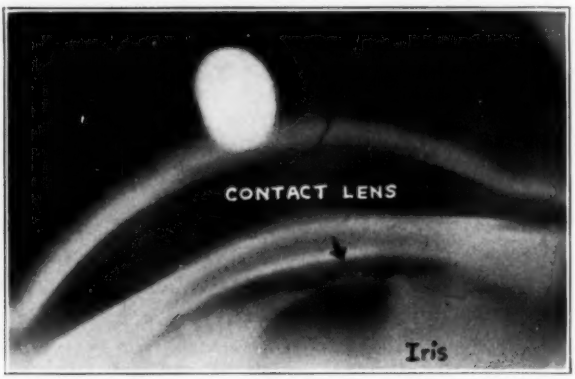

Fig. 11.—Angle at the root of a congenital coloboma of the iris (patient L. F.).

dermal tissue at the angle recess was noted in each. Anderson [8] attributed the glaucoma to persistence of the uveal meshwork or to adhesions between the stump of the iris and the cornea or the scleral meshwork.

(*d*) Congenital Coloboma of the Iris: Two cases of bilateral typical coloboma of the iris with evidence of glaucoma were studied gonioscopically. They may be briefly summarized as follows:

Case 1.—L. F., a 54 year old white man, was first seen on June 15, 1939, because of a history of failing vision and decreasing visual fields for one year. His corrected vision was 20/25 in the right eye and 20/40 in the left. The right visual field was constricted to within 15 degrees of fixation and the left to within 5 degrees of the fixation point. The tension was 48 mm. of mercury (Schiötz) in each eye and remained between 30 and 56 mm. in each eye even after administration of miotics. Both disks were excavated. Bilateral typical colobomas of the iris were present. The chamber angles were open in each eye except for the area of the coloboma, where a bridge of rudimentary iris was adherent to the trabecular wall (fig. 11). In each eye the number of ciliary processes in the area

of the coloboma was diminished. Those present appeared small and distorted. The intraocular tension in both eyes was well controlled by trephining operations.

CASE 2.—F. McM., a 54 year old white man, was first seen on Nov. 14, 1939, because of decreasing vision. He had bilateral colobomas of the iris and dense lenticular nuclear sclerosis in each eye. The right eye, with only light perception and projection, turned out 20 degrees. The right visual field was considerably constricted in its upper half as tested with a 5 mm. white object at 330 mm. The tension in the right eye varied between 40 and 26. The tension in the left eye remained normal. Both corneas were small, the right measuring 9 mm. and the left 10 mm. in the horizontal meridian. Gonioscopically the angles were shallow but open. Each eye had a rudimentary iris bridge at the base of the coloboma.

(*e*) Spherophakia: One eye of a 17 year old white girl with bilateral spherophakia revealed a definitely open angle in the lower portion in spite of a tension remaining constantly between 52 and 82 mm. of mercury (Schiötz) with both miotics and mydriatics. No evidence of vascular congestion was present. The other eye could not be examined because of poor cooperation.

It must be concluded that gonioscopically there is evidence of an anomaly of the angle in association with hydrophthalmia and aniridia but not in association with the other conditions discussed. This means, of course, not that anomalies may not be present but simply that they are not grossly visible.

POSTOPERATIVE GONIOSCOPIC OBSERVATIONS ON EYES WITH PRIMARY GLAUCOMA

The cause of the relatively frequent failure of operations for glaucoma to reduce the ocular hypertension to normal is usually a mystery to the operator unless gonioscopic examination is resorted to. The relation between the gonioscopic findings after over 300 such operations and their success or failure has been studied. In a third of these the eyes were examined both before and after operation.

1. *Iridectomy.*—Since von Graefe's introduction of iridectomy, many theories have been advanced to explain its action in relieving glaucoma, particularly the acute congestive type. Weber [20] expressed the belief that the operation reopens the closed chamber angle. Czermak [33] asserted that cutting off the pectinate ligament facilitates direct communication between the anterior chamber and Schlemm's canal. De Wecker [40] expressed the belief that by iridectomy a filtering scar is formed accidentally, with or without incorporation of uveal tissue. Smith [41] showed

40. de Wecker, L.: Traité théorique et pratique des maladies des yeux, Paris, A. Delahaye, 1867, vol. 2, p. 571.

41. Smith, P.: The Pathology and Treatment of Glaucoma, London, J. & A. Churchill, 1891.

in 1 case that a filtering cicatrix resulted after iridectomy. His description of the globe was as follows:

> The globe was bisected in the vertical meridian through the center of the coloboma. At this point the stump of the iris adheres to the cicatrix, but the latter is solid, and shows no channels in its substance. But at both extremities of the cicatrix the conditions are different; union is incomplete; iris-tissue is included in the cicatrix; and there are small open channels passing completely through the corneo-sclera and leading into an open space beneath the thickened and elevated conjunctiva. This condition is just what a minute examination of the cicatrix in many living eyes which have been cured by iridectomy would lead us to expect.

Troncoso,[25] with gonioscopic evidence, held that

> . . . reopening of the closed angle is not necessary for the success of an iridectomy in acute glaucoma. This operation succeeds in lowering the tension and in many cases improves vision even when the angle remains totally closed. The detachment of the peripheral synechiae from the cornea, although beneficial to the nutrition and function of the eye, is not a condition sine qua non for the reestablishment of the outflow even when a nonfiltering scar is present. In eyes with simple glaucoma and with the angle open, iridectomy may be successful in lowering high tension, although no change has been wrought in the condition of the angle.

Troncoso supported the idea that the operation works by "re-establishment of free communication between the anterior and posterior segments of the eye."

In a previous paper [1] I stated that the findings in a small group of cases of congestive glaucoma seemed to agree with the statements of Troncoso. I have since found sufficient evidence to change my opinion in regard to the action of iridectomy. From a study of 13 cases of acute congestive glaucoma in which the tension became normal with administration of miotics (table 1) it was evident that the reduction in tension was associated with opening of at least a portion of the circumference of the chamber angle. It therefore seemed logical that iridectomy must function in the same way, by freeing that portion of the angle where the iridectomy was done. However, on actual examination of 14 iridectomized eyes in which the tension became normal, 8 had partial opening of the angle and 6 had completely obliterated angles. Of those with partial opening of the angle, 2 had been subjected to true basal iridectomies, while in the other 6 the stump of the iris was plastered against the wall of the angle. In 5 of the 6 eyes with completely obliterated angles filtering cicatrices were believed to explain the normalization of tension. Small, ragged pieces of included iris tissue could be seen incarcerated in the operative incision and sometimes even projecting out under the conjunctiva. In 1 of the 5 a large filtering bleb was formed (fig. 12). A smaller bleb was present in a second eye, while in the others pressure on the globe through the lids produced small areas of edema of the conjunctiva. In the sixth case, in which the angle was completely closed,

the lens had been removed after the iridectomy, so that interpretation of the action of the iridectomy might have been equivocal. For that reason it was not considered.

In 5 of the 6 cases in which there were closed angles and normal tension, tests were made to determine the drop in tension on application of the new Schiötz tonometer with the 15.5 Gm. weight for two minutes, for comparison with that observed in 26 controls (over 50 years of age) with normal refraction (by a method modified from that of Böck, Kronfeld and Stough [41a]). In the 5 subjects the tension dropped 16 mm. (A. K.), 9.5 mm. (F. B.), 8.5 mm. (R. M.), 8.5 mm. (M. T.) and 7.5 mm. (C. E.), respectively. In the normal controls it dropped from 2.5

Fig. 12.—Filtering bleb following iridectomy for acute congestive glaucoma (patient A. K.).

to 6.5 mm., averaging 4.4 mm. In 2 successfully iridectomized patients with partly open angles it dropped only 3 mm. in each case (J. S. and L. S.), indicating absence of external filtration.

Six other eyes with acute congestive glaucoma were examined after unsuccessful iridectomy. All of these had completely obliterated angles, with plastering of the root of the iris against the wall of the angle in the area of operation.

In 1 case of chronic congestive glaucoma iridectomy resulted in reduction of the intraocular tension to normal and in partial opening of the angle.

41a. Böck, J.; Kronfeld, P. C., and Stough, J. T.: Effect on Intraocular Tension of Corneal Massage with the Tonometer of Schiötz, Arch. Ophth. **11**:797 (May) 1934.

Eleven eyes with chronic simple glaucoma on which iridectomy was done were examined postoperatively. Nine of these were absolutely glaucomatous eyes in which the stage of vascular decompensation had been reached. The operation was unsuccessful in the 2 eyes which did not have absolute glaucoma and in 7 of the 9 eyes with absolute glaucoma. In the other 2 eyes with absolute glaucoma the tension was reduced to normal. Both had filtering cicatrices.

A total of 32 iridectomized eyes were examined gonioscopically.

Success of the iridectomy was found to be inversely proportional to the length of time between the onset of the attack of acute glaucoma and

TABLE 6.—*Relation Between the Interval Before Iridectomy and the End Results of Operation*

Patient	Interval Between Onset of Attack and Iridectomy	Success or Failure in Reducing Tension to Normal	Angle After Operation
1. L. E.	2 months	Failure	Not seen
2. E. A. (right eye)	2 weeks	Failure	Not seen
3. A. J. (right eye)	3 weeks	Failure	Not seen
4. A. J. (left eye)	3 weeks	Failure	Not seen
5. R. H. (right eye)	3 weeks	Failure	Not seen
6. M. P.	11 days	Failure	Not seen
7. M. S.	9 days	Failure	Obliterated
8. K. K.	10 days	Success	Partly open
9. L. S.	4 days	Success	Partly open
10. E. A. (left eye)	2 days	Success	Not seen
11. F. B.	2 months	Success; filtering cicatrix	Obliterated
12. M. T.	2 months	Success; filtering cicatrix	Obliterated
13. A. M.	5 weeks	Success; filtering cicatrix	Obliterated
14. C. E.	2½ weeks	Success; filtering cicatrix	Obliterated
15. J. S.	3 weeks	Success	Partly open (basal iridectomy)
16. R. M.	2 weeks	Success; gross filtering cicatrix	Obliterated
17. A. K.	10 days	Success; gross filtering cicatrix	Obliterated

the time of iridectomy. If operation was done before dense adhesions had formed, part of the closed angle was opened and the tension reduced. If the adhesions had become too firm, the operation was unsuccessful, except when inclusion of the iris and a filtering cicatrix resulted. For this study 17 acutely glaucomatous eyes (table 6), including all those previously discussed concerning which the necessary information was available, and a number of iridectomized eyes in which the angle was not seen postoperatively were used. In 7 cases of unsuccessful operation the interval between the onset of the attack and the iridectomy ranged from two months to nine days (average three weeks). Of the 10 cases in which the operation was successful, the angle was partly open in 3 and could not be studied in a fourth. The intervals in the 3 cases in which the angles were partly open were three weeks, ten days and four days respectively. In the first case a true basal iridectomy had been

done. In the 6 other cases filtering cicatrices were present. In them the interval varied between two months and ten days (average, four and seven-tenths weeks).

The results in this small series and in cases of normalization of tension with miotics lead to the conclusion that iridectomy alone is the procedure of choice for acute glaucoma during the interval between attacks or after relief from an attack has been obtained with miotics. Early in an acute attack it would also serve to break the vicious combination of blocking of the angle and increasing pressure and would allow the angle to become patent again. Late in an attack, however, after firm adhesions have formed, perhaps a combination of iridectomy with partial iris inclusion might well be substituted for the classic operation. The ab externo approach also tends to insure incarceration of tags of iris in the wound and thereby promotes filtration.

These findings support the beliefs of Weber and de Wecker and may perhaps be best expressed by the statement of Fuchs [42] that "iridectomy acts primarily by opening the closed chamber angle, a thing which is possible only when the angle has not been permanently closed by exudation. After permanent closure by a peripheral synechia iridectomy can do good only in case it produces a filtering cicatrix."

2. *Cyclodialysis.*—Since Heine's [43] introduction of cyclodialysis as a surgical method of relieving glaucoma, many papers have been written concerning the mode of action of the operation. Heine himself expressed the belief that a communication between the anterior chamber and the suprachoroidea was the channel of drainage. Others held that the procedure acted by severing afferent arteries and injuring nerves, thus causing partial atrophy of the ciliary body and, subsequently, decreased formation of aqueous. Salus,[44] in a comprehensive paper, championed the latter idea. No definite evidence was submitted until Elschnig's [45] report in 1932 of the histologic picture of an eye on which a successful cyclodialysis had been performed fourteen years previously. He found an opening between the anterior chamber and the supraciliary space in the area of operation.

The first gonioscopic evidence of the mode of action of cyclodialysis was presented by Vannas [46] in 1935. He studied 37 cases of cyclodialysis and found patent clefts in 20 and none in 13. These findings were

42. Fuchs, E: Textbook of Ophthalmology, translated by A. Duane, ed. 7, Philadelphia, J. B. Lippincott Company, 1923, p. 799.

43. Heine: Die Zyklodialyse, eine neue Glaukomoperation, Deutsche med. Wchnschr. **21**:824, 1905.

44. Salus, R.: Die Zyklodialyse, Klin. Monatsbl. f. Augenh. **64**:433, 1920.

45. Elschnig, A.: Zur Werkungsweise der Zyklodialyse, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **49**:277, 1932.

46. Vannas, M.: Zykloskopische Untersuchungen über das Verhalten des Strahlenkörpers nach der Heinsichen Operation, Klin. Monatsbl. f. Augenh. **95**: 692, 1935.

uncertain in 4. In 30 of the 37 there was normal tension. Four of the eyes with open clefts had been operated on respectively eight, nine, ten and thirteen years before.

In 1936, Barkan [47] reported the gonioscopic data in 14 cases of primary glaucoma in which cyclodialysis had been done. In each of 11 cases in which the operation had been successful a patent cleft was found. In 3 cases in which it had been unsuccessful reattachment of the ciliary body had occurred. In 1 of the latter a second cyclodialysis was successful and resulted in the formation of a patent supraciliary space.

In a total of 65 cyclodialyses on eyes with primary glaucoma, I found an "all or none" relation between success of the operation and the formation of a communication between the anterior chamber and the supraciliary space. Of the 65 cyclodialyses, 34 (52.3 per cent) were

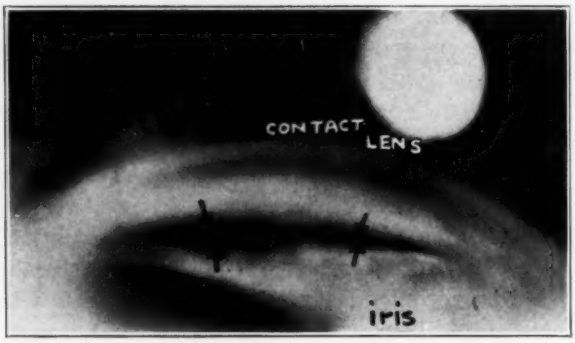

Fig. 13.—Successful cyclodialysis. Note the adhesion between the two portions of the open cleft.

successful in reducing the tension to normal. The tonometric tensions varied from 8 to 21 mm. of mercury (Schiötz), averaging 15 mm. in 33 cases. In 1 case the tension at times reached 32 mm. and so required miotics for control. In this case there was a very small patent cleft extending about 5 to 10 degrees of the circumference of the angle. In each of the 33 others there were patent clefts (figs. 13 and 10 *D*). The tension had no apparent relation to the breadth of the cleft in these cases. In the 31 cases of unsuccessful cyclodialysis no cleft formation was revealed. In nearly all there was stripping of a portion of Descemet's membrane, and in some there was reattachment of the ciliary body posterior to its original position. Occasionally one sees what appears superficially to be an open cleft, but closer examination reveals that it extends only a short distance and is a blind pocket. Transillumination of the sclera at this area helps determine this. In cases in which the cleft is broad, the lamina fusca of the sclera can be seen.

47. Barkan, O.: Mode of Action of Cyclodialysis, Am. J. Ophth. **19**:21, 1936.

Occasionally one finds another reason besides an open cleft for success of the cyclodialysis operation. In 1 case, observed after completion of this study, the tension remained normal in spite of no visible cyclodialysis cleft. Examination of the sclera at the site of the operative

Fig. 14.—Traumatic cyclodialysis in a patient with hypotony following injury.

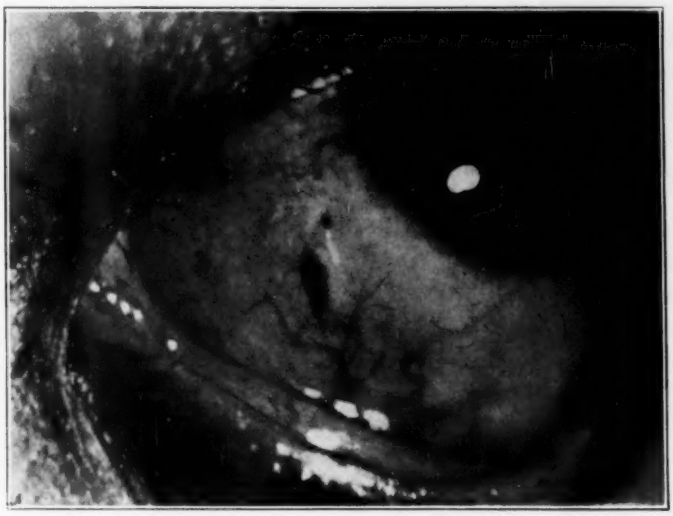

Fig. 15.—Filtering choroidal incarceration at the site of the cyclodialysis incision in an eye with a closed cyclodialysis cleft.

incision revealed an incarceration of choroid in the wound, with resultant subconjunctival filtration (fig. 15).

The presence of synechiae in the chamber angle before operation had little effect on the success of the operation. Twenty-four of the 65

eyes treated by cyclodialysis were observed preoperatively with the gonioscope. Seven had completely closed angles, and 4 had angles which were partly open. Functioning clefts were obtained in 5 of the 7 eyes with obliterated angles and in 2 of the 4 eyes with partly open angles.

Cyclodialysis for primary glaucoma is usually advocated only for the chronic simple type. Gradle [48] advocated the operation for "simple noninflammatory glaucoma without inflammatory reaction." An analysis of the series of 65 cases of cyclodialysis seems to bear this out. The operation was performed on 52 eyes with simple glaucoma, on 8 with acute congestive glaucoma and on 5 with chronic congestive glaucoma. Of the 8 with acute glaucoma, it was successful in 3 and unsuccessful in 5. In 1 of those in which success was obtained a sudden acute attack occurred nine months later, which required further surgical treatment.

Of the 5 cyclodialyses done on eyes with chronic congestive glaucoma, 3 were successful and 2 failed. One of the successfully treated eyes remained entirely normal for two years, after which the ocular hypertension recurred.

In none of the cases of successful cyclodialysis in the 52 eyes with simple glaucoma was there any recurrence of ocular hypertension following subsidence of all postoperative reaction, in spite of the fact that a number of these operations were done on eyes with shallow chambers. The longest interval between operation and observation in my series was five years.

Removal of a cataract from 1 eye on which cyclodialysis had been successfully performed resulted in closure of the cleft and later required repetition of the operation. The second operation also was successful.

It seems, then, that cyclodialysis probably is rather permanent in its effect in cases of simple glaucoma but, because of congestive episodes, may subsequently fail in cases of the congestive type of glaucoma and should probably not be used in the latter. It should probably not be used in eyes with simple glaucoma when further intraocular surgical procedures are contemplated.

In order to determine the length of time which may elapse after operation before one can judge the success or failure of cyclodialysis, I ascertained the time between operation and the first occurrence of tension above 28 mm. of mercury (Schiötz) in 19 eyes with simple glaucoma in which the cyclodialysis clefts had closed and the tension had become elevated. There were 9 instances of high tension on the patient's first return visit. In 2 instances each there were normal tensions for three, five and six weeks, respectively. In 1 each there were normal tensions for one, four, nine and sixteen weeks, respectively. In

48. Gradle, H. S.: Concerning Cyclodialysis in Simple Glaucoma, Am. J. Ophth. **14**:1101, 1931.

other words, for the eyes with normal tensions at the time of the first return visit the interval varied from one to sixteen weeks, averaging five and eight-tenths weeks.

Because of the interval necessary before one can determine success or failure tonometrically, gonioscopic study has been very helpful in determining the prognosis. The absence of any cleft a week or two after operation indicates that the operation has failed in spite of the fact that the tension may be low at the time of gonioscopic study.

The cause of failure of many well performed cyclodialyses is undoubtedly the persistence and organization of a blood clot in the cleft. I have observed the closure of a cleft by organization of such a clot in 1 case. To avoid this complication and to minimize trauma, I believe that a decrease in the extent of sweep of the cyclodialysis spatula, the avoidance of the horizontal meridian, where the long posterior ciliary vessels and nerves are located, and the use of physostigmine salicylate postoperatively to keep the cleft patent will help. Blaskovics'[49] method of cyclodialysis, advocated and used by Kronfeld, certainly is to be considered in this regard. Following Gradle's suggestion that the operation be done in one of the upper quadrants and that the patient be propped up in bed postoperatively will probably help to keep blood out of the cleft when hemorrhage occurs. Perhaps, also, Troncoso's magnesium implantation operation[50] may be an answer to this problem, though it has had, as yet, insufficient trial.

In addition to the 65 cyclodialyses performed for primary glaucoma, 56, which were done for secondary glaucoma following cataract operations were examined gonioscopically (see section on secondary glaucoma). The two groups were entirely alike in regard to the "all or none" relation between the formation of a channel to the supraciliary space and success of the operation, as well as in regard to the presence of a prognostically important interval without which one cannot judge, without use of the gonioscope, the success or failure of the operation.

3. *Elliot Trephining Operation.*—Werner,[27] in a study of 14 trephined eyes, found an open angle in 3 and large corneal synechiae, especially in the upper part, in the remaining 11. He concluded that synechiae which form after trephining have no significance so far as the action of the operation in reducing ocular hypertension is concerned, since the outflow is no longer dependent on natural channels.

McLean[28] concluded, from a series of 14 trephined eyes, that the relation between the presence or absence of synechiae and the success

49. Blaskovics, L.: Cyclodialysis Inversa, Szemészet **70**:5, 1935; abstracted, Zentralbl. f. d. ges. Ophth. **35**:455, 1935.

50. Troncoso, M. U.: Cyclodialysis with Insertion of a Metal Implant in the Treatment of Glaucoma, Arch. Ophth. **23**:270 (Feb.) 1940.

or failure of the operation is a direct one, similar to the relation between the two which he found in a large series of iridencleises.

I have attempted to study the various factors involved in successful trephining operations. Among these are the relation between preoperative synechiae and success of the operation, between postoperative synechiae and success, between patency of the trephine openings and success and between the rapidity of postoperative formation of the anterior chamber and success.

To determine the relation between preoperative synechiae and the success of trephining operations, 64 trephinings were studied both preoperatively and postoperatively (table 7). The angles in 52 eyes were originally open; those in 7 were partly open, and those in 5 were obliterated. The tension was reduced to normal in 67.3 per cent of the eyes with open angles, in 57 per cent of the eyes with partly open angles and in 40 per cent of the eyes with obliterated angles. Comparison of this small series of cases indicates a slight difference in the results. It is to be expected that eyes without preoperative synechiae should yield more favorable results, since the iris base is farther back. Consequently there is less danger of the opening being plugged by ciliary processes or by iris.

This same series of cases was studied to show the effect of operation on the formation of synechiae. Of the 52 angles which were open preoperatively, 2 (3.8 per cent) remained open, 34 (65.3 per cent) were partly closed and 16 (30.7 per cent) were entirely closed. Of the 7 angles which were partly open preoperatively, 5 remained unchanged and 2 became completely obliterated. The results corroborate previous evidence that trephining operations have a great tendency to cause formation of peripheral anterior synechiae. The same is true of all operative procedures after which the anterior chamber is collapsed for any considerable length of time.

To determine the relationship between the presence of postoperative synechiae and the success of the operation, the data on 90 trephinings seen only postoperatively were added to the postoperative findings in the group just considered. Table 8 indicates the total results. In order to use the combined statistics on the several types of glaucoma, the series was first studied to see whether trephining is equally successful for all types. Of the 154 trephinings, 15 were for acute congestive glaucoma, 16 for chronic congestive glaucoma, 118 for chronic simple glaucoma and 5 for absolute glaucoma. The tension was reduced to normal in 9 (60 per cent) of the eyes with acute congestive glaucoma, in 10 (62 per cent) of those with chronic congestive glaucoma, in 79 (66 per cent) of those with chronic simple glaucoma and in 3 (60 per cent) of those with absolute glaucoma. It is obvious that the operation may be equally effective for all types of glaucoma.

Results of a study of the relation between tension control and postoperative synechiae do not agree with the findings of McLean.[28] Of 101 trephined eyes in which the tension was reduced to normal (table 9),

TABLE 7.—*Relation Between Synechiae, Tension Control and Patency of Trephine Openings in Sixty-Four Trephined Eyes Seen Gonioscopically Both Preoperatively and Postoperatively*

Type of Glaucoma	Angle Before Operation	Tension	Angle After Operation	Patency of Trephine Opening
Acute congestive, 6	Open, 3	Normal, 2	Obliterated, 2	Patent, 1 Not seen, 1
		Uncontrolled, 1	Obliterated, 1	Plugged by iris, 1
	Partly open, 1	Controlled with miotics, 1	Partly open, 1	Patent, 1
	Obliterated, 2	Normal, 1	Obliterated, 1	Patent, 1
		Uncontrolled, 1	Obliterated, 1	Not seen, 1
Chronic congestive, 3	Open, 2	Normal, 2	Partly open, 1	Iris adherent to anterior lip, 1
			Obliterated, 1	Not seen, 1
	Partly open, 1	Normal, 1	Obliterated, 1	Not seen, 1
Chronic simple, 54	Open, 47	Normal, 31	Open, 1	Patent, 1
			Partly open, 21	Patent, 14 (3 partly filled with ciliary processes, 4 with iris, 1 with lens) Not seen, 6 Iris adherent to anterior lip, 1
			Obliterated, 9	Patent, 5 (1 partly filled with iris, 1 with sclera) Not seen, 4
		Controlled with miotics, 7	Partly open, 6	Patent, 4 (3 partly filled with iris, 1 with lens) Not seen, 2
			Obliterated, 1	Patent, 1 (partly filled with iris)
		Uncontrolled, 9	Open, 1	Patent, 1
			Partly open, 6	Patent, 1; bleb scarred Plugged, 5 (1 filled with ciliary processes, 4 with iris)
			Obliterated, 2	Plugged, 1 (filled with ciliary processes) Not seen, 1 (too far back)
	Partly open, 5	Normal, 3	Partly open, 2	Patent, 1 (partly filled with iris) Not seen, 1
			Obliterated, 1	Patent, 1
		Uncontrolled, 2	Partly open, 2	Plugged, 2 (filled with iris, ciliary process)
	Obliterated, 2	Controlled with miotics, 1	Obliterated, 1	Not seen, 1
		Uncontrolled, 1	Obliterated, 1	Not seen, 1
Absolute glaucoma (chronic simple), 1	Obliterated, 1	Normal, 1	Obliterated, 1	Patent, 1

the angle was open in 7 per cent, partly closed in 48 per cent and entirely closed in 46 per cent. Of 22 trephined eyes in which the tension was normalized with miotics, 4 per cent had open angles, 72 per cent had

TABLE 8.—*Relation Between Synechiae, Tension Control and Patency of Trephine Openings in a Total of One Hundred and Fifty-Four Trephined Eyes Examined Postoperatively*

Type of Glaucoma	Tension Control	Angle After Operation	Patency of Trephine Opening
Acute congestive, 15	Normal, 9	Partly open, 3	Not seen, 3
		Obliterated, 6	Patent, 3
			Not seen, 3
	Controlled with miotics, 1	Partly open, 1	Patent, 1
	Uncontrolled, 5	Obliterated, 5	Plugged, 3 (1 filled with ciliary body and iris, 1 with iris, 1 with lens)
			Not seen, 2
Chronic congestive, 16	Normal, 10	Partly open, 4	Patent, 1
			Not seen, 2
			Iris adherent to anterior lip, 1
		Obliterated, 6	Patent, 2
			Not seen, 3
			Late infection, 1
	Controlled with miotics, 3	Partly open, 3	Patent, 1 (partly filled with ciliary process)
			Not seen, 2
	Uncontrolled, 3	Partly open, 2	Plugged, 2 (1 filled with iris and ciliary process, 1 with iris)
		Obliterated, 1	Not seen, 1
Chronic simple, 118	Normal, 79	Open, 7	Patent, 2
			Not seen, 3
			Iris adherent to anterior lip, 2
		Partly open, 39	Patent, 23 (4 partly filled with ciliary processes, 5 with iris, 1 with lens)
			Not seen, 13
			Iris adherent to anterior lip, 3
		Obliterated, 33	Patent, 20 (1 partly filled with iris and ciliary process, 1 with iris, 5 with ciliary processes, 1 with sclera, 1 with lens)
			Not seen, 11
			Iris adherent to anterior lip, 1
			Lens capsule adherent to anterior lip, 1
	Controlled with miotics, 17	Open, 1	Connective tissue in trephine, 1
		Partly open, 12	Patent, 7 (4 partly filled with iris, 1 with ciliary process, 1 with lens)
			Not seen, 5
		Obliterated, 4	Patent, 2 (partly filled with iris)
			Not seen, 1
			Ciliary process adherent to anterior lip, 1
	Uncontrolled, 22	Open, 1	Patent; bleb scarred
		Partly open, 13	Patent, 1; bleb scarred
			Plugged, 11 (2 filled with iris and ciliary processes, 8 with iris, 1 with ciliary processes)
			Not seen, 1
		Obliterated, 8	Plugged, 3 (filled with ciliary processes)
			Not seen, 4
			Late infection, 1
Absolute glaucoma (chronic simple), 5	Normal, 3	Partly open, 2	Not seen, 2
		Obliterated, 1	Patent, 1
	Controlled with miotics, 1	Obliterated, 1	Not seen, 1
	Uncontrolled, 1	Obliterated, 1	Plugged by lens, 1

partly open angles and 22 per cent entirely closed angles. Of 31 eyes on which unsuccessful trephinings had been done, the angle was open in 3 per cent, partly closed in 48 per cent and obliterated in 48 per cent. Thus there seems to be no parallelism between the presence of postoperative synechiae and the success or failure of trephining. Another factor is obviously the important one, namely, patency of the trephine opening. The relation of this factor to the success of the operation is shown in table 10. Of a total of 86 eyes in which the trephine opening was visible, 52 had normal tensions; the tension in 12 was controlled with miotics and that in 22 was uncontrolled. In 28 per cent of the normalized eyes the trephine hole was partly filled by iris, ciliary proc-

TABLE 9.—*Relation Between Postoperative Synechiae and Tension Control in One Hundred and Fifty-Four Trephining Operations*

Tension	Angle After Operation		
	Open	Partly Open	Closed
Normal, 101	7 (7%)	48 (48%)	46 (46%)
Controlled with miotics, 22	1 (4%)	16 (72%)	5 (22%)
Uncontrolled, 31	1 (3%)	15 (48%)	15 (48%)
Total, 154	9	79	66

TABLE 10.—*Relation Between Tension Control and Patency of Trephine Openings*

Tension	Visibility of Trephine Opening		Visible Trephine Openings with Partial or Complete Plugging
	Not Seen	Seen	
Normal, 101	49	52	15 partly filled (28%)
Controlled with miotics, 22	10	12	10 partly filled (83%)
Uncontrolled, 31	9	22	20 completely plugged (90%)
Total, 154	68	86	45

esses, lens or sclera. In those in which the tension was controlled with miotics, 83 per cent of the trephine openings were partly filled with the same tissues. In addition, 1 opening contained connective tissue. In 22 cases in which the tension was uncontrolled, 20 openings (90 per cent) were completely plugged by iris, ciliary processes or lens. In the 2 remaining cases (tension uncontrolled) there were patent trephine openings but scarred, nonfiltering conjunctival coverings.

It seems logical to conclude that the chief cause of failure of trephining operations is plugging, partially or completely, of the trephine opening by iris, ciliary processes, both of these, lens or scleral tissue (figs. 10 *E*, 10 *F* and 16). Troncoso and Reese,[51] in 1933, reported the gonioscopic data on 29 trephined eyes. They found adhesions to the

51. Troncoso, M. U., and Reese, A. B.: Gonioscopic Findings After Elliot Operation, Tr. Ophth. Soc. U. Kingdom **53**:366, 1933.

sclera in 7, incarceration of the ciliary processes in 12 and exudates attached to the ciliary body and the sclera in 6. Seven had consecutive iritis, and only 3 showed normal healing. In my series 1 opening was partially filled and 4 were completely filled with both iris and ciliary processes. Twelve were partly filled and 10 completely filled with iris. Eleven were partly filled and 4 completely filled with ciliary processes. Three were partly filled and 1 completely plugged by knuckles of lens. One was partly filled with scleral tissue, and 1 was partly filled with connective tissue. In those in which the lens substance was present in the opening, no cataract formation was observed during more than a year of observation. McLean [28] made a similar observation in 1 case observed for two years.

Other factors which contribute toward failure of trephinings are the formation of peripheral anterior synechiae, previously discussed, and

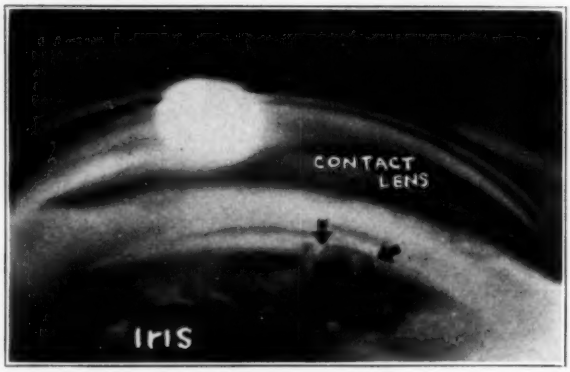

Fig. 16.—Partially filled trephine opening. Portions of the iris pillars and one ciliary process are adherent to the walls of the trephine opening.

the improper placing of the trephine opening. The first factor is important only when the trephine opening is completely or nearly completely plugged. Then the drainage from the eye is further embarrassed by the newly formed synechiae, and the intraocular tension may rise above its preoperative level.

Proper placing of the trephine opening requires knowledge of the anatomic fact that the sclera overlaps the cornea 2 mm. at the upper limbus, 1.5 mm. below and 1 mm. at the sides, so that a trephine opening placed properly above would probably be too far back at the sides. Similarly, if the angle is closed preoperatively, the trephine opening may enter the posterior chamber behind the area of iris adhesion and permit easy access of the ciliary processes to the trephine opening. Rapid release of aqueous when the bulging iris is opened at the time of operation may be one of the factors in causing forward prolapse of the ciliary processes into the trephine opening.

Among the causes of obscuration of the trephine opening gonioscopically, besides placing of the trephine opening too far back, are adhesions between the iris and the anterior lip of the trephine, adhesions between the anterior lens capsule and the anterior lip of the trephine and extremely shallow anterior chambers. The adhesions to the anterior lip of the trephine obviously occur during the postoperative period, while the anterior chamber is collapsed.

Because of incomplete records of the time of formation of the anterior chamber after many trephinings, the relation between the duration of collapse of the chamber and the success of the operation could be ascertained in only 20 cases. In 11 cases of unsuccessful trephinings the time required for formation of the anterior chamber varied up to eighteen days and averaged six and six-tenths days. In 9 cases of successful trephining it varied to twenty-one days, averaging six and seven-tenths days. There is probably no relation between the two. Again, the patency of the new drainage channel is probably the all-important factor.

4. *Iris Inclusion Operations (Iridotasis, Iridencleisis).*—Werner [27] studied 19 eyes on which iridencleisis had been performed, 18 with simple glaucoma and 1 with chronic congestive glaucoma. He saw the included iris gonioscopically in each case. Of 10 eyes with simple glaucoma in which the angle had been free preoperatively, it was entirely closed after the operation in 6. In 3 other instances of simple glaucoma with preoperative trabecular adhesions, 1 remained unchanged, and in 2 the adhesions progressed to become corneal.

McLean [28] recently reported 54 cases of iridencleises and stated the opinion that the postoperative increase of peripheral synechiae influenced the outcome, especially in borderline cases.

I studied a series of 13 iris inclusion operations to determine the relation between preoperative angle synechiae and the success of the operation (table 11). Nine cases in which there were open angles, 3 in which there were partly open angles and 1 in which there was an obliterated angle preoperatively were observed. In 6 of those with open angles the tension was reduced to normal. In 1 it was controlled with miotics, and in 2 it remained uncontrolled. In the eyes with preoperatively partly open angles all the operations were successful, as was that in the eye with the obliterated angle. No conclusions can be drawn from this small number of cases, but no relation between the presence of preoperative synechiae and failure of the operation is suggested.

A total of 48 eyes on which iris inclusion operations were performed (table 12) was studied to determine the relation between the success of the operation and the postoperative formation of synechiae. Of 33 eyes in which the tension was reduced to normal, the angles in 26 were partly open and those in 7 were obliterated. Of 6 in which it was controlled with miotics, the angles in 4 were partly open and those in 2 obliterated.

Of 9 in which it was uncontrolled, the angles in 5 were partly open and those in 4 obliterated. No parallelism between success or failure and postoperative synechiae was apparent.

The observations concerning these iris inclusion operations indicate that, while it is true that peripheral anterior synechiae add to the embarrassment of the previous channels of outflow, one must consider the

TABLE 11.—*Relation Between Synechiae and Tension Control in Forty-Eight Operations for Inclusion of the Iris*

Type of Glaucoma	Tension	Angle Before Operation	Angle After Operation
Acute congestive, 5	Normal, 1	.....	Partly open, 1
	Controlled with miotics, 1	.....	Obliterated, 1
	Uncontrolled, 3	.....	Obliterated, 3
Chronic congestive, 8	Normal, 6	Open, 1	Obliterated, 2 Partly open, 4
	Controlled with miotics, 2	Open, 1	Partly open, 1 Obliterated, 1
Chronic simple, 34	Normal, 25	Open, 4 Partly open, 2	Partly open, 20
		Open, 1 Obliterated, 1	Obliterated, 5
	Controlled with miotics, 3	.....	Partly open, 3
	Uncontrolled, 6	Open, 2	Partly open, 5 Obliterated, 1
Absolute (chronic simple), 1	Normal, 1	Partly open, 1	Partly open, 1
Total		13	48

TABLE 12.—*Relation Between Postoperative Synechiae and Tension Control in Forty-Eight Operations for Inclusion of the Iris*

Tension	Angle After Operation	
	Partly Open	Closed
Normal, 33	26	7
Controlled with miotics, 6	4	2
Uncontrolled, 9	5	4
Total, 48	35	13

patency of the new drainage channel at the same time. Unlike the trephining operations, in the iris inclusions the outflow channels are not often visible (fig. 17). Incarceration of the iris can be seen in every case, but the functional status usually has to be determined from the clinical findings. In a number of cases it appeared that normal function was most often associated with inclusion of a thick piece of iris in one edge of the wound, together with edema of the cornea at the wound site, and that failure was associated with inclusion of a thin, flat piece of iris tissue. It is probable that the pressure of the two lips of the wound on a flat piece hinders filtration, while if the iris is jammed into one corner of the incision a separation tends to remain between the lips

of the wound. Use of a scleral punch forceps to make a small sclerectomy will probably add to the success of the operation.[51a]

5. *La Grange Sclerectomy.*—Only 4 operations of this type were done. One, performed on a patient with acute congestive glaucoma with the angle open preoperatively, was entirely successful in reducing the tension to normal. There was a patent trephine-like opening in the eye, and the angle was partly open postoperatively. Three others were performed on patients with chronic simple glaucoma; the tension in 2 instances was controlled with miotics, and 1 operation was unsuccessful, although the tension remained normal for ten weeks postoperatively. The angle postoperatively was open in 1 and partly closed in the other 2. In 2, incarcerations of the ciliary body in the sclerectomy opening were observed.

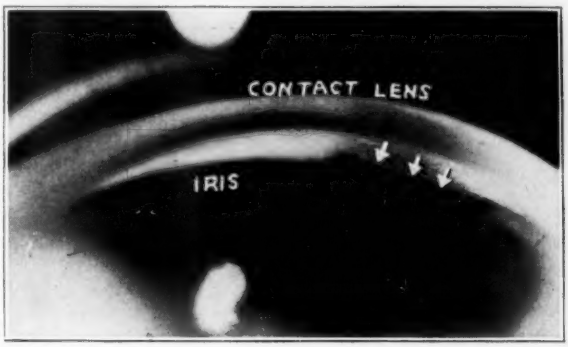

Fig. 17.—Incarceration of the iris in a case of successful iridencleisis. The arrows point to the keratome incision.

To prevent the formation of peripheral anterior synechiae in all types of filtering operations, the injection of air into the anterior chamber as advocated by MacMillan,[52] McLean [28] and others is probably the only direct means at the operator's disposal.

6. *Goniotomy.*—Since Barkan's [7] introduction of goniotomy or goniotrabeculotomy for the relief of the deep chamber angle type of simple glaucoma, only 1 report has appeared.[1] In that paper the results in a small series of 9 cases were described. The procedure used was that of Barkan except that in 6 of the cases (those of glaucoma capsulare) the operation was slightly modified by turning the Barkan knife sideways and curetting rather than incising the trabecula. In every case the tension remained normal for a period ranging from three days

51a. Greenwood, A.: Comparison of Operations for Chronic Glaucoma, Arch. Ophth. **10**:472 (Oct.) 1933.

52. MacMillan, J. A.: Air Injection as a Factor in Maintaining Filtration Following Corneoscleral Trephining in Glaucoma, Tr. Am. Ophth. Soc. **37**:127, 1939.

to four months but finally returned to its previous glaucomatous level. In 2 the tension remained normal for two and four months, respectively. In the latter case curettage had removed a portion of the trabecula and the scleral spur in one small area. This apparently allowed fluid to enter the supraciliary space and acted for a time exactly like a cyclodialysis. It was suggested that perhaps in the other cases the incision or the curettage had removed a small portion of the endothelial water barrier and had allowed the aqueous to infiltrate the ciliary body. There it would be absorbed by the blood. Or, perhaps, the trauma of operation caused reflex vasodilatation in the iris and increased vascular absorption of fluid by way of the capillary walls. At any rate, from the present knowledge of the physiology of the angle, it does not seem likely that incision of the trabecula or of Schlemm's canal has any but a temporary effect on the ocular tension.

7. *Vogt's Cyclodiathermy Operation.*—A series of operations of this type is being studied by Dr. S. J. Meyer at the Illinois Eye and Ear Infirmary. Cyclodiathermy has been used in 6 operations outside the series. Five of the 6 operations were unsuccessful in reducing the tension. The chamber angle in the area of application of the diathermy is usually obliterated or has trabecular adhesions, probably depending on the degree of reaction in the ciliary body.

GONIOPHOTOGRAPHY

For the teaching of gonioscopy, the use of color photographs is extremely helpful and often essential. I have obtained such photographs by a simple technic which is neither time consuming nor too expensive.

Thorburn,[26] in 1927, photographed the chamber angle of the human being, using a stereoscopic camera with a Gullstrand point-o-lite lamp. The minimal exposure was three to four seconds, and the magnification obtained was 1.7. A reproduction from Thorburn is in Castroviejo's paper on goniophotography.[53] In that paper (1935) Castroviejo presented a method of using the Leitz ultrapak with 3.8 objective, a no. 4 ocular and a Leica camera on a micro-ibso attachment. He used supersensitive panchromatic film at 1/20 second exposure. The magnification he obtained was 15. With such high magnification, the obliquely seen wall of the angle is not in good focus in its entire extent. The lack of color differentiation makes interpretation of the landmarks difficult.

The technic I have used depends on synchronized flash illumination and shutter release. A Kine-Exacta camera with a 2.8 Tessar lens is mounted on a gonioscopic stand or some other suitable carrier. A synchronized flashlight system [54] is used for illumination (fig. 18). In this system a no. 1 nonfrosted photoflood lamp is connected with a

53. Castroviejo, R.: Goniophotography, Am. J. Ophth. **18**:524, 1935.

54. This equipment was supplied by Barnett Laboratories, 160 North Wells Street, Chicago.

resistance so that the lamp gives an illumination of about 2 foot candles, sufficient for focusing the angle of the eye, to which a gonioscopic contact lens has been applied. As soon as the synchronizing switch is pressed, the resistance is shunted out of the circuit, and a momentary flash (about 175 foot candles) is obtained, synchronized with the release of the shutter. A standard exposure of 1/25 second at f2.8 is used.

Fig. 18.—Goniophotographic illuminating system.

The photoflood lamp is housed in a hooded goose-necked desk lamp and held about 4 to 5 inches (10 to 12 cm.) from the contact lens. Type A kodachrome film is used.

Dr. Harry S. Gradle made valuable suggestions and made this work possible. Dr. Peter C. Kronfeld suggested the tonometer massage tests referred to in the section on iridectomy. Dr. E. E. Grossmann assisted with these tests. Mr. Stanley Wolf aidid in copying the angle photographs.

Tenth Infantry Medical Detachment, Fort Custer, Mich.

Obituaries

ACHILLES EDWARD DAVIS, M.D.
1866-1941

Achilles Edward Davis died at his home in Scarsdale, N. Y., on Jan. 17, 1941. Dr. Davis was born at Harrodsburg, Ky., on Feb. 18, 1866. He was the son of Dr. H. C. Davis and Josephine LeCompte Davis. His grandfather as well as his father was a physician.

He attended Centre College at Danville, Ky., and received his bachelor's degree in 1886 and his master's degree in 1889. In the same year he received the degree of Doctor of Medicine from the University of Louisville. In 1891 he studied abroad, and in 1892 he began practicing in New York. He was consultant on diseases of the eye at the New York Post-Graduate Medical School and Hospital and consulting ophthalmic surgeon at the Manhattan State Hospital at Central Islip, L. I., after 1899; at the United Hospital at Port Chester, N. Y., after 1911, and at Ossining Hospital, Ossining, N. Y., after 1913.

He specialized in diseases of the eye and first attracted attention by a paper entitled "Light Streak on the Retinal Arteries." He published many other technical papers and books, among which were: "The Refraction of the Eye," published in 1900; "Handbook of the Anatomy and the Diseases of the Eye and Ear" (in collaboration), published in 1904, and "Medical Treatment of Cataract," published in 1937. He also wrote the section on refraction in the "American Encyclopedia of Ophthalmology," published in 1919. He was greatly interested in the medical treatment of cataract and devoted a great deal of time to the investigation of this subject. He had an abiding faith in medical methods of handling this condition.

Dr. Davis was always interested in military affairs and served as a captain in the World War. He was an ardent Democrat and a great admirer of Lincoln and of Pasteur. For recreation he indulged in bridge playing and amateur gardening. Occasionally he wrote for the daily papers on the latter subject.

He is survived by his wife, Mrs. Josephine Robinson Davis; three sons, Achilles E. Davis Jr., Lieut. Clay R. Davis, of the United States Army, and LeCompte K. Davis, and by his identical twin brother, Dr. George E. Davis, who is in practice in New York.

He was a member of the New York State Medical Society, the New York Ophthalmological Society, the New York Academy of Medicine, the American Medical Association, the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology and other medical organizations. He was also a member of the Kentuckians, the Huguenot Society of New York and the Lotus Club.

CLYDE E. MCDANNALD.

News and Notes

EDITED BY W. L. BENEDICT

GENERAL NEWS

Resignations and Appointments.—After serving for twelve years as a member of the Editorial Board of the ARCHIVES OF OPHTHALMOLOGY and for ten years as head of the department of ophthalmology at the Harvard Medical School and concurrently as ophthalmic chief of staff at the Massachusetts Eye and Ear Infirmary, Dr. J. Herbert Waite has recently resigned these positions.

Dr. David G. Cogan, acting director of the Howe Laboratory of Ophthalmology of Harvard University, will join the Editorial Board of the ARCHIVES OF OPHTHALMOLOGY.

Dr. Edwin B. Dunphy has been designated clinical professor of ophthalmology at Harvard University and ophthalmic chief of staff at the Massachusetts Eye and Ear Infirmary.

Section Meeting.—The Section on Ophthalmology and Otolaryngology of the Medical Society of the State of New York will hold its meeting on Wednesday, April 30, at 9 a. m., in the Iroquois Room of the Hotel Statler, Buffalo. The program consists of the following papers:

Instruction Hour: Tonometry
Jonas S. Friedenwald, M.D., Baltimore (by invitation)

1. Ophthalmoscopic Findings Versus Sinusitis
Arthur J. Bedell, M.D., Albany, N. Y.
2. Subnormal Vision and Occupation Aptitude
Albert C. Snell, M.D., Rochester, N. Y.
3. The General Practitioner's Share in the Campaign for the Prevention of Blindness in Patients Suffering from Glaucoma
Mark J. Schoenberg, M.D., New York

Examinations for Orthoptic Technicians.—The American Orthoptic Council will hold its oral and practical examinations for orthoptic technicians on Oct. 17, 1941, in Chicago. The written examinations will be held locally throughout the country on September 17. Application for permission to take the examinations, properly endorsed by a sponsor, must be received before September 1. These applications should be sent either to the American Orthopic Council, 23 East Seventy-Ninth Street, New York, or to Dr. E. B. Dunphy, 243 Charles Street, Boston.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

HYDROCARBON METABOLISM OF THE LENS: A SUMMARY OF SOME RECENT EXPERIMENTAL FINDINGS. R. WEEKERS, Ann. d'ocul. **177:** 118 (March) 1940.

A study of the lens metabolism has formed the greater part of ocular work in biochemistry. The ease with which this organ may be obtained, the excellent means of observation in vivo and the clinical interest in the subject of cataract form the incentive for this. In the last few years many observations have been made, but these represent only a small part of the complex changes occurring in the metabolism of a living organism. The different summaries reveal many gaps. To corroborate or refute the different hypotheses, Weekers undertook this study concerning the hydrocarbon metabolism of the lens.

The author reports under the following titles: 1. The Necessary Lens Power. 2. Lens Nutrition. 3. Permeability of the Capsule. 4. Hydrocarbon Metabolism of the Lens Parenchyma. 5. Oxygen-Reducing Systems. Each heading is discussed in detail. There is a long bibliography.

S. H. MCKEE.

Conjunctiva

SULPHONAMIDE TREATMENT OF BACTERIAL AND TRACHOMATOUS CONJUNCTIVITIS. A. F. MACCALLAN, Brit. M. J. **1:** 482 (March 23) 1940.

In an investigation of the influence of the new drugs of the sulfonamide group on trachoma, MacCallan emphasizes the importance of a detailed description of the conjunctival picture on which a diagnosis is based. He also states that it should be studied after the treatment has been stopped. He has used a derivative of sulfonamide known as sulfapyridine, giving 3 Gm. the first day, followed by 2 Gm. daily for nine days. He found that it might be necessary to repeat this course of therapy after an interval of ten days.

A case is reported in which there was in addition to the trachoma a heavy growth of pneumococci and hemolytic streptococci. These accidental infections as well as the blepharitis were cured, but the trachoma remained unaltered.

The author states that up to the present the drugs of the sulfonamide group have been found to be without effect on any virus disease, which would also include trachoma. The good results obtained in the treatment of trachoma have been produced by the elimination of superimposed bacterial infections.

ARNOLD KNAPP.

Comparative Ophthalmology

DEVELOPMENT AND DIFFERENTIATION OF THE TADPOLE EYE. A. GLUCKSMANN, Brit. J. Ophth. **24:** 153 (April) 1940.

The development of the tadpole eye and especially of the retina is described from the stage of the primary optic vesicle to that of the fully differentiated organ. Except for one stage of development, the number of mitotic cells in the tadpole retina remains remarkably constant, while the total cell count steadily increases. This suggests that only one of the daughter cells resulting from mitosis can divide again. Three distinct waves of cell degeneration are observed during the development of the retina. The first is due to the death of the dividing cells of the central part of the retina, and it is followed by the appearance of ganglion cells in this area. The second wave involves the resting cells of the future inner nuclear layer and precedes the differentiation of this layer, and the third is caused by the death of resting cells of the outer nuclear layer and occurs immediately before its appearance. The cells concerned in these waves of degeneration seem, therefore, to be those which are caught by the new differentiation impulse after they have already begun to obey the last.

Owing to the progressive decrease in cell size consequent on the decreasing yolk content, cell movements are unrestricted during the early development of the tadpole. After the division of the retina into a differentiated area and a peripheral germinative zone, cell movements are mostly confined to migration from the germinative zone into the inner nuclear layer and from this into the outer nuclear layer.

The importance of these cell activities in determining the course of the morphogenic changes which occur during the development of the amphibian retina is discussed, and an attempt is made to determine the origin of the factors which are responsible for the coordination of all cell activities into an orderly pattern which will insure the proper development of the eye as a whole.

W. ZENTMAYER.

Congenital Anomalies

MICROPHTHALMIA AND THE VISUAL PATHWAYS. A CASE ASSOCIATED WITH BLINDNESS AND IMBECILITY, AND SEX-LINKED. S. E. WHITNALL and R. M. NORMAN, Brit. J. Ophth, **24:** 229 (May) 1940.

The authors give the following summary of the case reported in their article:

"(1) A microphthalmic imbecile 17 years of age, with a unique ophthalmological family history, presented at autopsy special features in his eyeballs and visual tract.

(2) The eyeballs were completely disorganized and could never have transmitted light—equivalent to an anophthalmic condition.

(3) The effects of this deprivation of light impulses were reflected in a pronounced poorness of development of the optic nerves, chiasma, tracts and lateral geniculate bodies.

But beyond this region:

(4) The superior quadrigeminal bodies were quite normal in size and structure.

(5) The optic radiation was well formed, and its fibres were myelinated.

(6) The cortical visual areas, though their surface extent was much less than usual and one was grossly deformed, presented a normal lamina granulosa and a well marked stria of Gennari.

(9) The imbecile condition is possibly explained by an imperfect general cortical cellular composition; for there were no gross cerebral pathological lesions present."

The article is illustrated.

W. ZENTMAYER.

Cornea and Sclera

VITALITY OF THE CORNEA. P. BUSSE GRAWITZ, Arch. f. Ophth. **141:** 59, 1939.

Leber and Senftleben regarded dried corneas as dead. The cellular reactions which occurred in dried corneas on intraperitoneal implantation into rabbits were interpreted as invasion by leukocytes of the host. Filatoff and Bajenova, on the other hand, reported actual growth of the epithelial cells as well as of fibroblasts in dried cornea kept in tissue culture flasks for forty-eight hours. "Dried corneae are therefore not dead."

In the paper under review, experiments are reported in which corneas of rabbits were exposed to temperatures varying from 60 to 205 C. for thirty to forty-five minutes and then implanted in the peritoneal cavities of rabbits for two days. The histologic study of these corneas revealed that temperatures of 195 C. and up destroy all vital processes completely and irreversibly. No leukocytic invasion, however, occurred in these dead corneas. After exposures to temperatures up to 175 C. certain active cellular reactions occurred during the period of intraperitoneal implantation.

The author's description of these histologic reactions is unfortunately not very clear. The most essential feature apparently was the formation of new chromatin, either in the form of round nuclei or, in more severely damaged tissue, by the transformation of collagenous fibers into chromatin. The effect of chemical and other noxious agents was also studied.

The author concludes that the vitality of the cornea surpasses that of any other known tissue and that intraperitoneal implantation of tissues is a very promising method for studies of the vitality of normal, damaged and neoplastic tissues.

P. C. KRONFELD.

Experimental Pathology

GRENZ-RAY TREATMENT OF EXPERIMENTAL INFECTION OF THE CORNEA. E. GALLARDO, R. PFEIFFER and R. THOMPSON, Am. J. Ophth. **23:** 41 (Jan.) 1940.

After experiments, the authors conclude that lesions and scars produced by intracorneal injections of staphylococci in rabbits were definitely and consistently smaller in eyes treated by repeated applications of grenz rays after the infection than were those in the untreated eyes of the same animals.

W. S. REESE.

A Comparison Between the Reaction of the Lens and that of the Cornea to Infra-Red Rays. A. Bakker, Arch. f. Ophth. **141:** 180, 1939.

The explanted surviving corneas of rabbits remained clear on direct exposure to infra-red rays. If the irradiation was made through an explanted iris put directly on the cornea the latter became cloudy. If in another glass container a surviving lens was put in front of the vessel containing the iris and the cornea so that the lens had to bear the brunt of the infra-red rays, only the portion of the cornea covered by iris showed any changes attributable to the irradiation. These experiments were undertaken in order to disprove Vogt's concept of the selective absorption of infra-red rays by the lens. P. C. Kronfeld.

General Diseases

Dysostosis Multiplex with Special Reference to Ocular Findings. S. J. Meyer and H. B. Akner, Am. J. Ophth. **22:** 713 (July) 1939.

Meyer and Akner report a case of dysostosis multiplex in a girl aged 7 whose mother noted that the child had a large abdomen, flexion and stiffness of the fourth finger and spinal curvature at the age of 1½ years. After an attack of mumps when she was 3 years of age it was noticed that the child could not see well. A complete account of the physical and laboratory data is given, including roentgenograms of the skull, spine and extremities. The eyes showed marked cloudiness of the corneas, each of which measured 14 mm. in diameter. Corneal sensibility and ocular tension were normal. Biomicroscopic examination revealed diffusely scattered throughout the stroma and Bowman's membrane many small punctate grayish white opacities. The authors discuss dysostosis multiplex and its differential diagnosis. They note that this case was unique in that megalocornea was present. W. S. Reese.

Alterations in the Fundus and Histologic Examination of the Eye in Cases of Tuberculous Sclerosis (Maladie de Bourneville). K. Hirose and R. Nagae, Ann. d'ocul. **176:** 1 (Jan.) 1940.

This article deals with examination of the eye of a man aged 23 years, who in his infancy had many attacks of convulsions followed by poor physical development. At autopsy there was typical tuberculous sclerosis. Death was primarily due to cerebral hemorrhage. The patient, as had been noted during life, had ptosis of the right lid and total rigidity of the pupil. In addition to the loss of sight in the right eye, the papillary stasis was accompanied with several hemorrhagic areas in the right fundus. Histologic examination showed that this was not true papillary stasis. It was also established that there were solitary tuberculous tumors in the beds of the nerve fibers and hemorrhagic areas in each of the retinas.

The histologic examination is reported in detail, and the author concludes that the tumors of the papilla must be considered as alterations produced by the same causes that engendered the changes in the other organs. A bibliography is presented. S. H. McKee.

General Pathology

Nature of the Inclusion Bodies of Prowazek. Y. Sugita, Arch. f. Ophth. **141:** 46, 1939.

If a drop of a strong inorganic acid is introduced into an epithelial cell by means of a capillary pipet mounted on the micromanipulator, a well circumscribed disk-shaped portion of protoplasm is precipitated in the form of fine granules. If the specimen is then stained with Giemsa's solution the area where the precipitation has taken place resembles closely an inclusion body of the type first described by Halberstaedter and Prowazek. Because of the similarity between these trachomatous cell inclusions and the products of localized acid precipitation the author concludes that the granules of the Prowazek bodies also represent coagulated cell protein. P. C. Kronfeld.

Glaucoma

The Need for Social Service Work in Glaucoma. A. F. Jensen and H. S. Gradle, Am. J. Ophth. **22:** 993 (Sept.) 1939.

Jensen and Gradle review the records of 239 patients with glaucoma admitted to the Illinois Eye and Ear Infirmary. They tabulated the cases as to age, bilateral involvement, vision and length of time between the first symptoms and the first visit to the clinic. They found that a high percentage of patients failed to return for subsequent treatment and concluded that follow-up social service would remedy this.

W. S. Reese.

Inverse Cyclodialysis of Blaskovics. R. F. Pereira, Arch. de oftal. de Buenos Aires **14:** 547 (July) 1939.

After a brief review of the late Professor Blaskovics' work, Pereira reports a case of an inverse cyclodialysis performed according to Blaskovics' method, in which the spatula is introduced into the suprachoroidal space through a vertical incision made just below the insertion of the superior rectus muscle, with subsequent insertion into the anterior chamber. The patient suffered from chronic glaucoma and had been operated on a year before for retinal detachment by electrocoagulation, with a resulting vision limited to ability to count fingers at 2 meters. After the cyclodialysis vision was improved. C. E. Finlay.

Hygiene, Sociology, Education and History

A Study of Ocular Defects Among University Students. E. A. Thacker, Am. J. Ophth. **22:** 1003 (Sept.) 1939.

Thacker gives the following summary of his study of University of Illinois students.

"1. Of the male student body at the University of Illinois 34.3 percent have defective vision as determined by the Snellen test.

"2. Although it is admitted that the correction of muscle disorders may have necessitated some blurring at a distance, nevertheless the glasses of 30.2 percent of the students with defective vision did not correct the distance vision to normal.

"3. The visual acuity of 38.2 percent of the students with defective vision became worse since their entrance to the university. Of the sophomores, juniors, seniors and graduate students, respectively, 31.1, 37.5, 43.6, and 51.2 percent showed an increase in their visual defect.

"4. The greatest percentage of students with an increase in visual defect since entrance occurred in the Graduate School, the Physical Education, Fine and Applied Arts, Commerce, and Engineering Colleges, respectively.

"5. Evidence is produced showing that excessive use of the eyes is definitely a factor in aggravating an existing visual defect.

"6. Foci of infection in many cases cause abnormal vision or aggravate an already existing condition.

"7. The relationship of some of the common childhood diseases to ocular disorders is discussed.

"8. All students with defective vision should seek consultation concerning their eyes once yearly.

"9. Symptoms produced by errors of refraction are discussed. Many times defective vision is present without producing any recognizable subjective symptoms. Fifteen percent of the students with errors of refraction had no symptoms.

"10. The errors of refraction are classified according to the degree of defect.

"11. The types of ocular disorders affecting vision are tabulated.

"12. The cost of examination and glasses averaged $15.75, although there is quite a large range in costs. There was very little difference between the average charges made by the ophthalmologist and optometrist (not more than 10 percent)."

W. S. REESE.

Injuries

A CASE OF LUXATIO BULBI. E. C. ZORAB and W. L. BURNS, Brit. J. Ophth. **24:** 286 (June) 1940.

The case reported was a "black-out" casualty. A man, three quarters of an hour prior to examination, had struck his face on what he believed to be a biscuit tin. The left eyeball was dislocated anteriorly, and the lids were almost closed behind the eyeball. There was a large conjunctival tear with a subconjunctival hemorrhage. The cornea was completely denuded of epithelium. None of the muscles appeared to be ruptured. Vision was limited to light perception. The fundus could not be seen in detail. Block of the left facial nerve at the neck of the mandible was made with a 4 per cent solution of procaine hydrochloride. Almost immediately vision improved. The exophthalmos subsided, and the lids closed over the eye. The outcome was full recovery, with vision 6/5.

W. ZENTMAYER.

DYSFUNCTION OF THE HYPOPHYSIS DUE TO FRACTURE OF THE SELLA TURCICA. MARIOTH, Arch. f. Ophth. **141:** 188, 1939.

As the result of a collision while riding a motorcycle, a youth 19 years of age sustained a fracture of the skull extending into the floor of the sella. The ocular findings shortly after the injury were palsy of both sixth nerves, extensive bleeding into all four lids and under the bulbar conjunctivas and bilateral traumatic retinopathy (Purtscher). When the patient regained consciousness a defect in the nasal half of the left visual field was detected. Later, symptoms of acromegaly developed.

P. C. KRONFELD.

Lens

EXPERIENCES WITH LACARRERE'S ELECTRO-DIAPHAKIA. JESS, Klin. Monatsbl. f. Augenh. **103:** 99 (July) 1939.

After employing Lacarrere's method of extracting cataracts by electrocoagulation Jess simplified this method. He found that it is the more useful the more nuclear sclerosis is present. Hard brown cataracts yield the best results. Rupture of the capsule of the lens is a frequent source of disappointment with this operation. Its effect, on the other hand, is gratifying in the removal of dislocated lenses or cataracts or in cases of threatening dislocation during cataract extractions as reported by Elschnig. Publications of recent papers and opinions on modifications of this method are cited.

K. L. STOLL.

LENTICULAR REGENERATION (FORMATION OF LENTOIDS) IN SECONDARY CATARACT IN A FAMILY WITH HEREDITARY PERINUCLEAR CATARACT. F. RIEDL, Klin. Monatsbl. f. Augenh. **103:** 169 (Aug.) 1939.

In an exhaustive paper the author reports on a family with hereditary perinuclear cataracts in five generations. Eight members were afflicted, 5 of them in the last two generations; their cataracts had been extracted elsewhere. Detailed case histories are given. The eyes operated on showed peculiar spheroid, irregularly shaped or conic formations in the secondary cataracts, and 3 of these patients had minute yellowish or brownish globular formations on the iris. Similar bodies, forming clusters, were noted in the corneal operative scars of 2 patients and in the vitreous of another. No histologic relation existed between these formations and the secondary cataracts. The number of these bodies varied between fifty and one hundred in the divers eyes. A number of photomicrographs prove the similarity with normal lens fibers. These lentoids originated from the epithelial cells of the lens. It is possible that the lentoids on the secondary cataract and on the surface of the iris originated from epithelial lenticular cells which escaped into the anterior chamber during discission. It may be, on the other hand, that these epithelial cells became detached from the secondary cataracts after several years, as the lentoids were discovered several years after discission. Formation of lentoids appears to occur rarely. Only Thiel and Handmann have published papers on this phenomenon.

K. L. STOLL.

ZONULAR CATARACT AND SPASMOPHILIA. E. GSCHEIDEL, Klin. Monatsbl. f. Augenh. **103:** 194 (Aug.) 1939.

The author examined at the Children's Hospital of the University of Tübingen 70 children who had been treated previously for spasmophilia and whose ages ranged between 1 and 17 years. He found the phosphorus content of the blood normal or slightly lowered and the calcium content lower than normal. Of the 140 lenses, 77 were perfectly clear; in the others, dotted, linear or feathery opacities were noted in the cortex. Opacities on the posterior pole of the lens occurred in 1 child, anterior embryonal cataract in 3 and linear opacities in the nucleus in 3. Zonular cataract was discovered in none of these 70 children with manifest spasmophilia, who had been under clinical observation for long periods. The author, therefore, concludes that spasmophilia has no influence on the formation of zonular cataract.

K. L. STOLL.

Neurology

DIAGNOSIS, LOCALIZATION AND TREATMENT OF INTRACRANIAL SACCULAR ANEURYSMS. M. N. WALSH, Arch. Neurol. & Psychiat. **44:** 671 (Sept.) 1940.

The only characteristic syndrome produced by intracranial aneurysms is that which is associated with an aneurysm in the cavernous sinus below the bifurcation of the internal carotid artery. This was described by Foix as a syndrome of the lateral wall of the cavernous sinus. It consists essentially of pain and often anesthesia in the distribution of the first muscles supplied by the fourth and fifth cranial nerves and sometimes exophthalmos. A bruit is rare. Evidence of compression of the optic nerves, chiasm or tracts may be present, and after rupture changes in the visual fields the characteristic of interruption of the optic radiation may be found if there is extensive damage to the brain. Palsy of the third nerve alone, however, is not of localizing significance, as it may result from aneurysms situated on any of the basilar arteries, even outside the circle of Willis. Next to aneurysms located in the cavernous sinus in frequency of production of cranial nerve palsies are those at the junction of the posterior communicating artery and the internal carotid artery. It is estimated that 47 per cent of such aneurysms will give rise to palsy of the third nerve. Aneurysms below the bifurcation of the internal carotid artery are referred to as infraclinoid aneursysms.

R. IRVINE.

VASONEUROSIS WITH EYE-SYMPTOMS. BÉLA BOROS, Brit. J. Ophth. **24:** 179 (April) 1940.

A man 32 years old complained that in opening the eyes in the morning he often felt pains in them and they became injected. Repeated, similar and more frequent attacks occurred. During one such attack the right eyelids were swollen; there were photophobia and conjunctival and ciliary injection. Touching the eyeball caused great pain. Deep in the lower part of the cornea a slight haze was present. There were a few unusually thick nerves; the iris was slightly hyperemic. Tension in this eye was 15 mm. of mercury and in the left eye 21 mm. Vision in

both eyes was 6/6. All the symptoms "vanished before our very eyes." The patient had migraine, as did his mother and grandfather. He was neurasthenic and showed a vivid demographism. Undoubtedly the essence of the condition was vasoneurosis involving lessening of the blood vessel tone.

The author is inclined to consider the case as one of a variety of ophthalmomalacia rather than one of intermittent corneal edema.

W. ZENTMAYER.

Ocular Muscles

CONVERGENCE DEFICIENCY. I. MANN, Brit. J. Ophth. **24:** 373 (Aug.) 1940.

Mann states that she is not dealing with the subject of heterophoria as a whole but discussing a small group of cases characterized by the fact that there was deviation of 6 degrees or more (exophorial) on the Maddox wing test and that when ordered to converge voluntarily without an object to fix on the patients were totally unable to perform the movement even when it had been explained and demonstrated to them. The last-mentioned sign is considered of greater importance than the readings with the Maddox wing and the Maddox rod. The subjects showed a deviation of 6 to 22 degrees with the wing test. With the rod test they showed orthophoria, exophoria or esophoria for distance. The majority of them showed very low grade hypermetropic astigmatism.

There are two cortical areas (frontal and occipital) which are involved in ocular movements: The frontal area appears to govern voluntary movements, and if it is destroyed the patient cannot look in a certain direction on command, though he can follow a moving object, since the fixation reflex is mediated by the intact occipital center. If the occipital center is destroyed the fixation reflex is lost, though the patient can perform voluntary movements of the eyes. In cases like those under consideration it is likely that therapy should be directed at increasing the permeability of the frontal path by "facilitation," using conscious attention as the stimulus and the fixation reflex as the source of the impulse.

As to treatment, the first method includes graduated exercises in fusion on the synoptophore; exercises with prisms to produce convergence for distance, and exercises with a stereoscope and figures of graduated spacing and difficulty. The second method consists of the education of the patient in the appreciation of physiologic diplopia and the control of this; exercises with the small diploscope, and exercises in the fusion of streoscopic cards without a stereoscope.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

XEROPHTHALMIA AMONG CHILDREN IN BATAVIA. H. DE HAAS, J. H. POSTHUMA and O. MEULEMANS, Geneesk. tijdschr. v. Nederl.-Indië **80:** 917 (April 9) 1940.

According to de Haas and his collaborators, 259, or nearly 5 per cent, of the children admitted to the Central Civil Hospital in Batavia during

the period from 1935 to 1938 had xerophthalmia. More than 40 per cent of the children were either totally blind or blind in one eye. Disorders of the digestive tract occupied a prominent place among the diseases coincident with xerophthalmia, and it is probable that inadequate resorption and insufficient diet play an important part in the pathogenesis of xerophthalmia. Feeding with sweetened skimmed milk was responsible for xerophthalmia in two thirds of the total number of cases in which the patients were children less than 2 years old. An advanced state of dystrophy existed in 75 per cent of the children with xerophthalmia. The vitamin A content of the blood was lower in children with xerophthalmia than in those without it. The use of vitamin A and the continued oral administration of cod liver oil are recommended for the treatment of xerophthalmia, particularly during the early stages. Prophylaxis is more important than treatment; it should be directed toward improvement of the nutrition of the natives, particularly of children of preschool age.

J. A. M. A.

Pharmacology

VARIATIONS IN RETINAL AND HUMERAL BLOOD PRESSURE RESULTING FROM ACETYLCHOLINE AND SHORT WAVE THERAPY IN NORMAL AND HYPERTENSIVE SUBJECTS. M. CORRADO, Ann. di ottal. e clin. ocul. **68:** 1 (Jan.) 1940.

The literature indicates that acetylcholine produces a constant dilatation of the retinal arteries, while its effect on the retinal blood pressure is transient and variable.

In 23 normal subjects the retinal blood pressure was measured with the Bailliart dynamometer, while at the same time the intraocular tension and the humeral blood pressure were recorded. Then short wave therapy with a 6 meter apparatus was given for ten to fifteen minutes, the electrodes being placed before the eye and at the neck. After this the records were reported at intervals. After five or six days 0.2 Gm. of acetylcholine was injected subcutaneously, and the same data were recorded. In all but 4 subjects of this group, after injection of acetylcholine the retinal vessels showed dilation which began a few minutes after injection and continued for a variable period. The diastolic retinal pressure showed a slight reduction, seldom more than 5 Gm. on the Bailliart scale in 8 cases, no change being noted in the others. In 2 cases there was a slight increase in pressure, which was succeeded by normal pressure. After short wave therapy, vasodilatation was observed in 15 cases and a slight decrease in retinal blood pressure in 11.

For 22 subjects with arterial hypertension the same records were made. The effect of short wave therapy on humeral blood pressure in these patients was in most cases nil. The retinal blood pressure showed a reduction in 17 cases of between 10 and 20 Gm., which persisted from several hours to several days. Moderate vasodilatation occurred in only 8 cases. Acetylcholine produced reduction of humeral blood pressure in 12 cases and of retinal blood pressure in 9. Vasodilatation, on the other hand, was seen in 18 of 22 cases.

Short wave therapy, in other words, produced a more constant effect on retinal blood pressure, while the effect of acetylcholine was more

constant on vasodilatation, the two conditions being in many cases independent of each other. The effect of short wave therapy on the retinal blood pressure is considered by the author to be due to an effect on the hypothalamus, in which a special center for regulating the cerebral and hence the retinal circulation has been shown to exist. A combination of this effect with the direct vasodilatation caused by acetylcholine might in some cases be of advantage. A bibliography is included.

S. R. GIFFORD.

Refraction and Accommodation

CHROMATIC REFRACTION TEST APPLIED TO NEAR VISION. F. ROESSLER, Arch. f. Ophth. **141:** 305, 1939.

In 1927 Roessler published a subjective method for the estimation of astigmatism which was based on the phenomenon of chromatic aberration. Through a violet glass filter which absorbed the yellow and green rays and through a diaphragm 8 mm. wide the examinee viewed a strong light source from a distance of 175 cm. The subjective image of this diaphragm depended characteristically on the position of the examinee's retina with regard to the images produced by the red and blue rays. In the presence of astigmatism the image of the diaphragm was characteristically distorted. According to the author, the position and magnitude of astigmatism could easily be determined with this method. In about 50 per cent of unselected cases the result of the chromatic aberration test was identical with that of a careful manifest refraction. In the paper under review Roessler reports studies of near vision with his chromatic test. In eyes which were unable to accommodate, the difference between distance and near correction (both determined with the chromatic test) corresponded exactly to the difference in the position of the light source in the two tests. If the examinee's eye was capable of accommodation, its refractive power was found to have increased somewhat during the near test but not to the extent necessary for the distinct perception of reading matter at the distance at which the chromatic test was done. In other words, under the condition of this test the eye accommodated insufficiently. This is understandable, because the retinal images under the conditions of the chromatic test are fairly distinct irrespective of the amount of accommodation.

With the chromatic test the well known fact was confirmed that the amount as well as the axis of the astigmatism prevailing under conditions of near vision may or may not be the same as that prevailing under conditions of distance vision. The patients in whom the astigmatism remained the same for distance and for near vision were comfortable with their full cylindric correction. Patients in whom the astigmatism for distance was different from that for near vision were more comfortable if the smallest cylinder compatible with fair visual acuity was ordered.

In presbyopes from the age of 52 on the chromatic near test proved to be the quickest method of determining the proper correction for work at close range.

P. C. KRONFELD.

Retina and Optic Nerve

A Case of So-Called Oguchi's Disease in the U. S. A. B. A. Klien, Am. J. Ophth. **22:** 953 (Sept.) 1939.

Klien reports the case of a 44 year old woman who complained of night blindness which she remembered as having been of the same degree since infancy. Corrected vision in the right eye was 0.4 and in the left eye 0.8 — 3. In both fundi there was a wavy yellowish white discoloration surrounding the disks and maculas and most intense above and temporal from the maculas, extending well into the periphery, where a gradual transition into normal fundus with a distinctly visible choroidal pattern could be observed. The opacity was beneath the retinal vessels. After occlusion of the eyes for seven hours the opacity had disappeared completely. The fields were normal except for slight enlargement of the blind spots. The patient was completely blind for red and green and also had a severe defect in perception of blue and yellow. Klien discusses the disease especially from the etiologic standpoint.

W. S. Reese.

Retinal Hemorrhages After Transfusion. R. J. Gray, Am. J. Ophth. **22:** 979 (Sept.) 1939.

After a study of 85 cases totaling 343 transfusions, Gray draws the following conclusions:

"1. A definite etiological relationship of transfusions to retinal hemorrhages cannot be stated with certainty at the present time.

"2. Retinal hemorrhages following transfusions have occurred only in those cases in which a preexisting condition itself predisposed to them.

"3. An etiological role, if any, is certainly minor in action.

"4. Further investigation and observation must be carried out in this phase.

"5. The occurrence of retinal hemorrhages is not a serious complication although Schaly considered the opposite to be true."

W. S. Reese.

Panophthalmitis with Yellow Staphylococcus After Operation for Retinal Detachment. E. Hartmann, Bull. Soc. d'opht. de Paris **51:** 233 (April) 1939.

A 40 year old woman with myopia of 4 D. had visual difficulty with the right eye for two or three days. A detachment with a tear was found in the superior temporal quadrant. Following galvanocautery, a perfect result was recorded after two weeks. Three weeks later the retina in the superior nasal quadrant was found to be pale, and operation was done in this region. Fever and a rapid pulse were noted twenty-four hours after the operation. Iodine was given, as it was thought the patient had symptoms of hyperthyroidism. On the first examination of the eye after operation there was slight edema of the conjunctiva, but the vitreous was clear and the retina in place. Pain persisted in the eye. One week

later signs of endophthalmitis were marked. Heroic measures were without avail.

The author draws attention to several important facts: (1) the slow appearance of intraocular infection, with general symptoms; (2) the period of latency and the localization of the infection, and (3) the possibilities of intraocular infection due to procedures for reattachment of the retina.

L. L. MAYER.

RELATION BETWEEN INDIRECT TRAUMA AND RETINAL DETACHMENT. J. SCHAUREN, Arch. f. Ophth. **141:** 72, 1939.

By indirect traumas the author means slight accidents which lead to mild concussion of the head or of the entire body. Such concussions are entailed not only in accidents but in riding on a train or in a motor car and in walking downstairs. Physical strains, if they are definitely greater than the strain entailed in the usual work of the patient, have, in court and insurance cases, also been considered indirect traumas.

Schauren reviews the theories of the pathogenesis of retinal detachment with special reference to the question of indirect traumas. Leber denied their significance altogether, whereas Gonin, Vogt, Lindner and others regarded them as precipitating factors which are effective (that is, tear producing) only in eyes with preexisting pathologic changes. Valid conclusions with regard to the part played by these traumas cannot be drawn from the theories on the pathogenesis of retinal detachment. Valuable evidence is, however, provided by studies on the incidence of retinal detachment in (1) persons who have sustained severe concussions and (2) in laborers (soldiers, athletes, etc.) whose regular work constantly entails indirect traumas. In these two groups of persons retinal detachment is, according to the literature, very rare and certainly not more common than in laborers whose work does not entail any indirect traumas. The author's concept of retinal detachment is as follows: It is a disease entity which occasionally occurs in young persons but becomes more frequent with increasing age. The retinal tear and the retinal detachment are the symptoms which are recognized most easily with the ordinary clinical methods. Actually, the retinal tear and the retinal detachment are only links (though very important ones) in a long chain of pathologic events in the inner ocular membranes. An important factor in the formation of tears and in the development of the detachment is the tendency of the diseased retina to contract (Arruga). The influence of ocular movements on the size of the tear and the extent of the detachment is admitted. Indirect traumas cannot be regarded as actual causes of retinal detachment.

P. C. KRONFELD.

THE DEVELOPMENT OF THE THROMBOSIS OF THE CENTRAL VEIN [OF THE RETINA]. E. SEIDEL, Arch. f. Ophth. **141:** 151, 1939.

In a woman 45 years of age Seidel observed the development of thrombosis of the central vein of the retina from the time of the first subjective symptoms to the usual outcome, enucleation because of secondary glaucoma. The patient, apparently Seidel's office assistant, one morning suddenly complained of impairment of vision in the right

eye. Examination at this time revealed marked contraction of the retinal arteries, slight dilation of the veins, normal vision and a normal visual field. Since the patient had been having severe attacks of migraine, Seidel interpreted the narrowing of the retinal arteries as a symptom of an impending attack of migraine. Twenty minutes later the vision of the right eye was reduced to 20/200; the arteries were still contracted, but the veins were much wider and the disk hyperemic. Fine hemorrhages developed on the disk, and the peripapillary portion of the retina became edematous. On the following day the picture was essentially unchanged. Within the next week the veins became still more engorged; extensive hemorrhages appeared in the periphery, and the vision dropped to finger counting at 0.5 meter. Cardiovascular examination revealed normal conditions except for a systolic blood pressure of 150 mm. Despite the use of amyl nitrite and acetylcholine, secondary glaucoma set in two months later, necessitating enucleation. Pathologically, a thrombus extending from the lamina cribrosa back about 1 mm. was found in the central vein. The retinal arteries on the disk showed marked arteriolosclerosis (hyaline degeneration of the walls).

Seidel interprets the clinical and pathologic observations as follows: In the presence of a definite vasoneurosis (migraine) a spasm of the central artery occurred by which the pressure in the central vein was markedly lowered. Under the influence of the intraocular pressure plus the effect of the thickened artery the vein was compressed in the lamina cribrosa; it collapsed, and a thrombus formed in it. The sudden arterial ischemia was thus the chief factor in the development of the thrombosis. This view was first expressed by Theodore Leber.

P. C. KRONFELD.

Trachoma

THE TRACHOMA CLINIC AND ITS FUNCTION WITH ASSOCIATED DISEASES. E. CORNET, Ann. d'ocul. **176:** 849 (Dec.) 1939.

Trachoma is an ocular disease which has been known since antiquity, and it is regrettable that the question of its cause is still being investigated. Busacca, Cuenod and Natif and Poleff tend to attribute this disease to rickettsia-like elements, and, while these investigations are encouraging, one cannot accept such a theory without further thorough study of the question.

Thorough clinical examination remains indispensable. For centuries this disease has been diagnosed only by symptoms, and the symptomatology has remained confused. From numerous articles the writer concludes that many of the symptoms of trachoma are caused by other diseases grafted on it. If one should take the trouble to separate the clinical elements that belong to each of these different aspects, the clinical examination would be made much simpler.

The author's description is divided into (1) pure trachoma; (2) parainfections, and (3) a complicating trachoma. A definite classification of each is given and treatment suggested. A bibliography is presented.

S. H. MCKEE.

PARATRACHOMA. G. LUGOSSY, Arch. f. Ophth. **141:** 234, 1939.

Paratrachoma, or trachoma of genital origin, is the term advocated by Lindner for inclusion conjunctivitis of the newborn child and of the adult. Of this condition the author reports 2 cases, the patients being a newborn infant and its mother. At first only the child showed the picture of bilateral inclusion conjunctivitis. Later the mother's eyes also became affected, the infection having occurred by transfer of secretion either from the child's eyes to the mother's eyes or from the mother's vagina to her eyes. The literature on inclusion conjunctivitis is reviewed.

Conjunctival scars are not a part of the characteristic picture of inclusion conjunctivitis (or that of gonococcic conjunctivitis). During the first month inclusion conjunctivitis cannot be distinguished from true trachoma. After the first month predominance of hypertrophy in the region of the lower fornix is characteristic of inclusion conjunctivitis.

P. C. KRONFELD.

Tumors

MIXED-CELL TUMOR OF THE LACRIMAL SAC. J. L. MCCOOL, Am. J. Ophth. **22:** 734 (July) 1939.

McCool reports the case of a 54 year old man who complained of tearing and a mass in the region of the left lacrimal sac. He had noticed the lacrimation 6 months previously and a few months later had become conscious of a small lump over the site of the lacrimal sac. This was increasing in size. An attempt to pass a solution through the drainage apparatus failed, and on inspection a mass about the size of a small cherry was noted at the site of the lacrimal sac. The overlying skin was normal in color and not particularly tense. Palpation revealed a small, almost bony hard mass, the overlying skin being freely movable. The mass was extirpated and found to be a mixed cell tumor of the lacrimal sac. McCool comments on the rarity of such tumors of the lacrimal sac and reviews the literature, citing 10 cases in detail. W. S. REESE.

SARCOMA OF THE CHOROID DIAGNOSED AFTER INTERVENTION FOR DETACHMENT OF THE RETINA. P. VEIL, Bull. Soc. d'opht. de Paris **51:** 229 (April) 1939.

A woman aged 52 presented herself for an operation for detachment of the retina of the right eye. The onset was sudden and recent. The detachment was nasal and superior. The ocular tension was normal, but the visual acuity was reduced to 1/10. Rest in bed for forty-eight hours did not change the aspect of the detachment. Operative intervention with galvanocautery gave no indication of a tumor. Forty-eight hours after operation the detachment was generalized, with vision reduced to light perception. By transillumination a more or less opaque mass was noted in the region where the retina had been originally detached. Enucleation was decided on, and microscopic examination revealed sarcoma of the choroid. Although the tumor had to be classified as melanosarcoma, there were many areas entirely free of pigment.

L. L. MAYER.

Uvea

A Case of the Syndrome of Harada: Severe Uveitis with Bilateral Detachment and Encephalitis. Magitot and Dubois-Poulsen, Bull. Soc. d'opht. de Paris **51:** 223 (April) 1939.

A disease due to an unknown virus is described by Japanese authors, especially Harada. The syndrome consists essentially of bilateral uveitis causing rapid and complete detachment of the retina and the choroid. Encephalitic phenomena, dysacousia, delirium, vomiting, troubles of the hair system and leukoderma may accompany the condition. The authors report on a patient of 28 years whose symptoms occurred in three phases: (1) uveitis and detachment of both retinas; (2) encephalitic and meningeal symptoms, and (3) recuperation, with reapplication of the detached retinas and disappearance of the cerebral phenomena.

Hypotheses have been brought forth to the effect that the exciting cause of the condition may be tuberculous or that it may be due to a herpes virus.

L. L. Mayer.

Society Transactions

EDITED BY W. L. BENEDICT

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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Dec. 19, 1940

A Case of Adie's Syndrome. DR. THOMAS H. COWAN.

A case of Adie's syndrome with tonic reactions of the muscles of the iris and ciliary body is presented.

J. K., a man aged 31, was first seen in September 1940. He complained of poor distant vision and desired to have refraction performed. He stated on questioning that he was and had been in excellent general health, with no neurologic complaints. There was no history of syphilis, tuberculosis or any other condition.

The ocular examination revealed the following abnormalities: The right pupil was larger than the left and was slightly flattened above; there was little or no reaction to light when the eye was tested by the usual method of flashing a light into it, and the consensual reflex was absent. However, on prolonged exposure to light, that is, exposure for several minutes, a slight contraction of the pupil was noted. After removal of the stimulus the pupil slowly dilated. The direct and consensual reflexes of the left eye were prompt and of normal amplitude.

On accommodation the right pupil reacted well after a short delay, and on the strongest accommodation it contracted even more markedly than the left, the measurements being 2.5 mm. and 3.5 mm. respectively at the near point of accommodation. On relaxation of the accommodation the right pupil remained small for several minutes and then slowly dilated to its former size. The reactions of the left pupil during accommodation seemed entirely normal.

Careful measurements revealed that the right eye accommodated 12 D., its near point being about 8 cm., while the left accommodated 9 D., with a near point of 11 cm. Furthermore, in the right eye a spastic condition of the ciliary muscle similar to that in the iris was found, for the near point of 8 cm. was achieved much more slowly than was the near point of the left eye, and immediately on removal of the stimulus for accommodation the vision for distance was blurred in the right eye but slowly became clear again.

Accommodation in the left eye, however, occurred in a perfectly normal manner.

With administration of 5 drops of 4 per cent homatropine solution at fifteen minute intervals, the diameter of the right pupil was 5 mm. and that of the left pupil 6 mm. Refraction gave the following results:

Right eye, — 1.50 D. sph. ⌒ — 3.00 D. cyl., axis 20; vision, 6/6 +
Left eye, — 2.00 D. sph. ⌒ — 2.50 D. cyl., axis 160; vision, 6/6 +

Full correction was ordered and was worn by the patient in comfort for both near and distant vision in spite of the bizarre accommodative behavior.

The patellar reflexes were tested, but no reaction could be elicited; the patient stated that they had never been present. He also had noted the difference in size of the pupils and said that this had been present as long as he could remember.

Multiple Sclerosis as an Ophthalmoneurologic Condition. DR. WALTER I. LILLIE.

Multiple sclerosis is a rare disease characterized by intermittent involvement of the central nervous system and producing motor or sensory disturbances, the most common of which are involvement of the extremities and visual disturbances, such as diplopia, nystagmus and retrobulbar and optic neuritis. Contrary to textbook descriptions, retrobulbar neuritis and optic neuritis are among the earliest and most characteristic episodes in this disease. Of 500 definitely proved cases, visual disturbances constituted the first symptom in 15 per cent; in an additional 40 per cent visual manifestations represented the second or the third episode in the course of the disease. Ophthalmologically in cases of acute retrobulbar neuritis associated with multiple sclerosis one finds normal fundi, diminished vision and central scotomas. With severe involvement the central scotomas may extend to the periphery of the visual field or may even produce complete loss of vision. Pallor of the disk is a later sign. Optic neuritis is distinguished from retrobulbar neuritis in that the inflammatory process is at the optic disk and is visible with the ophthalmoscope. The prognosis for improvement is better with retrobulbar than with optic neuritis. The more common type of retrobulbar neuritis is acute, with sudden loss of vision, but there is also a chronic form, with insidious loss of vision.

The significance of retrobulbar neuritis is not thoroughly appreciated, as is evidenced by confusion in the literature and by the fact that most of the patients have been subjected to unwarranted types of therapy. It is imperative that such patients have complete general and neurologic examinations, including analysis of the spinal fluid. In addition to multiple sclerosis, the clinical syndrome of retrobulbar neuritis may be due to meningoencephalitis, basofrontal tumor, diabetes, pernicious anemia, leukemia, nicotine, alcohol, heavy metals and chemicals, lactation, sinusitis or hereditary disease.

The prognosis of retrobulbar neuritis is good, and in most cases vision returns to normal without treatment. This fact accounts for the unwarranted enthusiasm of the exponents of the various types of ther-

apy. This is particularly true of intranasal surgical treatment. My experience has been that sinusitis rarely if ever produces retrobulbar neuritis. Complete studies by the various consultants produce the best diagnostic and therapeutic results. Foreign protein therapy and pilocarpine sweats have proved of definite benefit in diminishing the course of the disease and in minimizing the residual functional impairment of the visual apparatus.

DISCUSSION

DR. MANUEL SALL: Retrobulbar neuritis as an early or associated symptom of multiple sclerosis is not uncommon. The syndrome tends to occur early in the course of the disease and is usually unilateral. Some idea as to its frequency may be obtained from the following figures: To sum up individual reports by Fleisher, Marburg and Weil, a total of 88 patients with retrobulbar neuritis presented signs and symptoms of multiple sclerosis. Adie, who studied 70 patients with acute retrobulbar neuritis during the first week of their illness, found that 31 presented signs of multiple sclerosis. It is probable that more patients would have shown evidence of multiple sclerosis had they been followed further. The figure obtained by Sachs and Friedman in 1921 as to the presence of retrobulbar neuritis in cases of multiple sclerosis seems rather low. Of 141 cases of multiple sclerosis, they found temporal pallor of the optic disk in 46; in 11 of these 46 there was optic neuritis. They made no mention of having performed studies of the visual fields or of the visual acuity in this series. The visual fields and the visual acuity are of far greater import in recognizing retrobulbar neuritis than are the appearance of the fundus and the pallor of the disks. The interval from the time of appearance of the optic neuritis to that of other signs of multiple sclerosis is usually not long. However, some rather rare cases have been reported, 1 case in particular, described by Kinnier Wilson. A 21 year old woman who presented the picture of retrobulbar neuritis had had no other symptoms for twenty-one years, when there developed spastic paraplegia and other signs of multiple sclerosis. It may be interesting to mention that nystagmus, the commonest symptom of multiple sclerosis, occurs in over three quarters of the cases. So-called "jelly nystagmus" has been described by Paton and is observed through the ophthalmoscope as a minute rapid movement of the eyeball; it is supposedly pathognomonic of multiple sclerosis. It occurs very infrequently. Argyll Robertson pupil is rare, as are pupillary differences and irregularities. From the point of view of the neurologist, it is interesting to note that the presence of lesions immediately behind the nerve head may give rise to edema of the disk that is hardly distinguishable from the true papilledema of increased intracranial pressure. The question of tumor of the brain presents itself. However, the fact that visual impairment has preceded the change in the fundus, the tendency of the edema to clear up rapidly and the absence of other evidence of increased intracranial pressure will exclude the diagnosis of tumor.

DR. JOSEPH C. YASKIN: Dr. Lillie has raised the important question of the cause of retrobulbar neuritis in relation to multiple sclerosis. There can be no disagreement that multiple sclerosis is the commonest

cause of retrobulbar neuritis. He brought up the question of the advisability of investigating and operating on the sinuses. This is an old quarrel between the specialist in diseases of the nose and throat, the neurologist and the ophthalmologist. From the practical standpoint, all that is known about multiple sclerosis is a little about its pathologic picture. Nothing is known about its pathogenesis or about its cause. It is not even known whether it is an inflammatory or a degenerative disease. Given, therefore, a grave ocular condition and the prospects of other complications, and given, further, clinical signs of marked sinus disease, it has been my feeling that the sinuses should receive appropriate attention. While the mere roentgen indications of thickened mucosa is not sufficient justification for opening the posterior sinuses, in the presence of retrobulbar neuritis it would be my feeling that the patient should be given the benefit of surgical intervention. Perhaps this is the focus that is responsible for the multiple sclerosis. Or is it not possible that the cleaning out of the sinuses will delay the development of further complications, at least for some time?

Dr. Maxwell Langdon: I think that Dr. Lillie was on very firm ground when he laid a great deal of stress on studying thoroughly and completely, from a broad standpoint, every case of retrobulbar neuritis. It is very difficult to determine the cause, and only by thorough and complete studies of all the possibilities can one hope to arrive at a just conclusion. Dr. Yaskin expressed the opinion that multiple sclerosis is the main cause of retrobulbar neuritis. Apparently he thinks that retrobulbar neuritis in the great majority of cases is associated with multiple sclerosis. I am not sure whether that is correct. Toxic amblyopia is almost as frequent a cause. Of course, the neurologist and the ophthalmologist attack the problem from distinctly different angles. The neurologist is interested in the multiple sclerosis; the retrobulbar neuritis is merely a phase, a part of the general picture. The ophthalmologist, on the other hand, regards the retrobulbar neuritis as the primary problem, and what he seeks is the cause of this neuritis. I think, therefore, that the neurologist would naturally ascribe retrobulbar neuritis to multiple sclerosis oftener than to any other cause. He would not encounter as many cases of toxic amblyopia. Dr. Yaskin also referred to sinus disease as a possible cause of retrobulbar neuritis. If retrobulbar neuritis is caused by sinus disease, it is so caused in a very small percentage of cases, possibly only a fraction of 1 per cent; I feel, however, that there are occasional cases of retrobulbar neuritis due to sinusitis. I do not agree with Dr. Yaskin that the sinuses should not be invaded unless free pus is found. In my own experience sinusitis which causes ocular disease is not that which is associated with free pus. It is very low hyperplastic inflammation of the sinuses, and it is difficult for the rhinologist to be sure that active sinusitis is present.

Dr. Francis H. Adler: There are two things about this disease that are curious. One is its predilection for such a localized part of the ocular pathways when it causes such a widespread and diffuse pathologic

process in the other parts of the central nervous system. I believe that every one agrees that multiple sclerosis singles out the papillomacular bundle in the optic nerve. The second point of interest is that in nearly all cases, even when the condition is subsequently proved to have been multiple sclerosis, improvement occurs after the sinuses are opened. It seems quite possible that some day it will be shown that opening the sinuses accomplishes more than mere drainage. It may well be that such an operation does something with reflex control of the local blood vessels which temporarily benefits the pathway. It is best to be pragmatic about treatment; if it is found that opening the sinuses helps, my feeling is that they should be opened.

DR. WALTER I. LILLIE: I knew that my pointed statements concerning sinusitis as an infrequent cause of retrobulbar neuritis would stimulate discussion. For the past fifteen to twenty years operations on the sinuses have been performed frequently for acute retrobulbar neuritis even when clinical and roentgen examination revealed little or no pathologic change. The clinical term hyperplastic sinusitis was given to this type of sinusitis to differentiate it from the classic purulent type.

The treatment of acute retrobulbar neuritis is not specific, and many methods are employed. Fortunately for the patient, the course of acute retrobulbar neuritis is self limited, persisting usually for ten days to six weeks. The prognosis for return of normal vision and visual fields is excellent, as only 2 per cent of the patients have a permanent visual defect. When such a defect occurs, I believe that the optic nerve has been affected in the bony optic canal, which impedes swelling of the nerve, so that pressure causes early damage. This can be compared to Bell's palsy; when the facial nerve is involved in the bony canal, permanent damage results. In 98 per cent of cases of acute retrobulbar neuritis the vision returns to normal regardless of the treatment employed. One may remove an appendix, a great toe or tonsils, aerate the sinuses or treat the posterior orbit with roentgen rays, as described recently in the French literature, and regardless of the method used, improvement in the retrobulbar neuritis will occur. My own experience with sinusitis as a causative factor is on the negative side. I once examined 200 patients with purulent or hyperplastic sinusitis, and not one had retrobulbar neuritis. All physicians, I am sure, would have to search their private and clinical case files thoroughly before they could find a patient with the syndrome of retrobulbar neuritis who was referred by a rhinologist. Dr. Adler has suggested that congestion following the sinus operation may be the factor determining resultant improvement. The same can be said for foreign protein therapy; the dilatation of the peripheral arterioles, rather than the amount of fever produced, is the probable reason for the good results obtained.

Retrobulbar neuritis associated with multiple sclerosis usually occurs during the fall and spring, suggesting an infection or a virus as a cause. It is my opinion that operative measures have no proper place in the treatment of acute retrobulbar neuritis.

Late Results of Resection of Cervical Sympathetic Ganglions in Cases of Retinitis Pigmentosa. DR. FRANCIS C. GRANT.

(A brief review of retinitis pigmentosa was given, and the possibility of its being due to interference with the blood supply to the retina and the choroid was suggested. The sympathetic nerve supply to the eye was reviewed and the operations proposed for denervation outlined.

Nine cases of retinitis pigmentosa were presented. All the patients were operated on by the "high operation," stripping of the common internal and external carotid vessels plus excision of the upper and middle cervical sympathetic ganglions and the intervening nerve. Constriction of the field with consequent loss of vision seemed definitely slower in the surgically treated patients than in those who were not operated on. Whether this operation is the best is questionable, but it seems the easiest and the safest. As the vertebral nerve is not sectioned, this may be a source of return of control by the sympathetic nervous system. Regeneration of nerves should not occur, as the operation is apparently postganglionic.)

DISCUSSION

DR. EDMUND B. SPAETH: So far as these cases of retinitis pigmentosa are concerned, there are two or three things that I can add to Dr. Grant's presentation from my standpoint. I collaborated with him in the handling of these patients. In the selection of the patients for operation we tried to match each patient selected with another, similarly affected one from the various clinics at our command and from our private practice, so that every surgically treated patient had as a running mate a patient who was not operated on. It is rather interesting that the running mates of some of the surgically treated patients have done almost as well, but not all quite as well as those operated on; given certain definite signs and indications, some of the surgically treated patients have done better than the others.

Two things are certain. The first is the appearance of the vessels before and after the operation. After the operation vessels appear within the threshold visibility of the ophthalmoscope which formerly were not present. To illustrate this point, I shall describe two sketches made in 1 of the cases presented. They are rough sketches made with an ophthalmoscope before and after the operation. Each vessel as seen with a Gullstrand ophthalmoscope was plotted out on each of the nerve heads and lettered to indicate whether it was an arteriole or a venule, and then the sketch was carefully dated. From time to time, at certain intervals, the same fundus was thoroughly studied. The second sketch was made eleven months later. The new blood vessel formations were sketched in with red. It was seen that in the final analysis there had occurred only arteriolar dilatation of an extent which permitted ophthalmoscopic visualization of the new vessels at this time. At no time did we find new venules; the new blood vessels were all arterioles.

The second observation, which I think is rather interesting, is the tremendous variability in the fields of vision for several months after

the operation. Dr. Evans made a comment applicable to this: "The fields dilate and recede, as if the tide were washing up on the sand and then receding, and they do this for several months after the surgery." With the same amount of illumination and the same size of stimulus, the field varies and changes. A recession, however, is never quite as marked as it was before the widening, though the field seems finally to become stabilized. As Dr. Grant said, we merely present our findings and permit others to draw their own conclusions.

DR. ISAAC TASSMAN: Dr. Grant touched on the question of the cause of retinitis pigmentosa. He stated that in 1 case it was congenital and that in the others the cause was unknown. I think that it is generally accepted that this is in most cases classified as a heredofamilial disease and is considered to represent a degeneration of the tapetoretinal type. It may occur in more than one member of the same family and in successive generations of the same family, although it does not necessarily do so. I therefore believe that it would be interesting to know whether there were any other cases in the families of these patients. In other words, were they typical in that respect? One may assume that the ophthalmic picture is characteristic, and one knows that in most cases of retinitis pigmentosa the deposits of pigment occur at the equator. The marked narrowing and attenuation of the retinal vessels leads to the question whether this is an associated abnormality or the cause of the degeneration. It is reasonable to expect that as a result of the sympathectomy there will be improvement in the vision, such as was demonstrated in these cases, but I should like to ask whether one can expect the same result in cases of this kind as in cases of essential hypertension, in which there is no pigmentary degeneration of the retina of this characteristic type.

DR. WALTER I. LILLIE: I should like to ask Dr. Spaeth, as the rationale of a sympathectomy is to obtain dilatation of the peripheral arterioles, whether the retinal arterioles were measured with a graticule before and after the operations. Also, were the cases classified as to the duration of the condition or the age of the patient? The best results from symphathectomy in cases of Raynaud's disease occur in those patients who obtain a dilatation of the peripheral arterioles with foreign protein injected intravenouly.

DR. MAXWELL LANGDON: I should like to ask Dr. Spaeth whether the dilatation of the vessels was of long duration. It seems to me that he reported some cases a year or so ago; he said that dilatation was obtained for a while and then there was regression.

DR. GEORGE D. GAMMON: I should like to ask Dr. Grant whether any regeneration occurs. It is well known that sympathetic fibers regenerate rather rapidly when cut postganglionically. The operation described is a rather limited sympathectomy of the carotid artery. In this type of operation the surgeon would possibly be interrupting postganglionic fibers in some cases and removing the ganglion itself in others. If there is any evidence of regeneration, might not this account for the variation in the results in different patients?

DR. FRANCIS H. ADLER: Would it not be better to do some of these operations on one side only, so as to have a comparison between the operatively treated side and the other? I should like also to ask whether the peripheral operation performed by Dr. Grant gives rise to a sensitization of the blood vessels to circulating epinephrine. If the operation were a central denervation, this peripheral sensitization would not occur.

DR. FRANCIS C. GRANT: I was afraid that somebody was going to ask me about the pathologic aspects of the condition, as Dr. Tassman did. Duke-Elder has presented a complete set of notes, to which I cannot add much more. Dr. Lillie asked about typhoid; it has been used as a method of treatment but, I imagine, has been abandoned. The number of treatments that have been used and abandoned is provocative. Of the two articles I ran across on this subject, one was in Dutch and the other in Russian.

As to Dr. Adler's question about operating on one eye and observing the other, that was done to a certain extent, because the patients were operated on at intervals of about six weeks to permit observation and comparison. The curious thing about it is that in about one-half the cases in which operation was performed on one side the effect was bilateral. I think that this is being recognized by those who operate in such cases. If one does a sympathectomy on the right side, one may obtain an increase in both fields. Every one who has done it has remarked this. One would have to follow the patient for three or four years to see what difference occurs in the second eye. This is a perfectly possible and logical thing to do. In a way it was done in this series, although we operated on the good eye, and the bad eye was usually very bad, so that there was not much comparison. Dr. Gammon has brought up the question of regeneration of the sympathetic nervous system and the question whether we perform a preganglionic or a postganglionic procedure. If the operation is postganglionic we are perhaps making the sympathetically innervated vessels in the retina somewhat more responsive to circulating epinephrine, as in the case of Raynaud's disease; the reason for the failure in cases of vascular disease of the upper extremity in the past has been that the postganglionic type of operation was carried through, and in consequence of that the vessels were more sensitive to the epinephrine and quasi recurrence took place. Regeneration is also more likely to occur after a postganglionic operative procedure. I am not quite clear whether we have done the preganglionic or the postganglionic operation. It is my opinion that we have done the postganglionic procedure. I think that in order to do the preganglionic operation one would have to become involved in the anterior route. My knowledge on this point is not sufficient to enable me to give an accurate answer. The regression is due to one of two things—to regeneration or to the configuration of the disease (if our findings are admitted).

Diagnostic Significance of Retraction of the Upper Lid. Dr. Francis H. Adler and Dr. Harold Scheie.

This paper will be published in full, with discussion, in a later issue of the Archives.

The Oculomotor Nerve: Clinicoanatomic and Etiologic Considerations. Dr. Joseph C. Yaskin.

The most common causes of paralysis of muscles supplied by the third nerve include syphilis, vascular lesions, trauma, diabetes, neoplastic processes, acute suppurative and nonsuppurative infections of the nervous system, chronic degenerative diseases, exogenous intoxications, botulism, mushroom poisoning and myasthenia gravis. An etiologic and anatomic diagnosis is at times difficult but is often necessary for therapeutic reasons. Such a diagnosis may depend on intimate knowledge of the course and relation of the oculomotor complex. This presentation attempts to discuss the various clinical types of oculomotor palsy from a regional standpoint and is summarized in the following outline.

Intraorbital Lesions of the Third Nerve

1. Intrinsic disease of muscles (abiotrophy; myopathy; thyrotoxicosis)
2. Myasthenia gravis
3. Trauma
4. Causes producing exophthalmos
   - (*a*) Causes originating in the orbit
   - (*b*) Causes operating by contiguity into the orbit
   - (*c*) Systemic and distal causes

Lesions of the Third Nerve in the Region of the Cavernous Sinus, Sphenoid Fissure and Middle Cranial Fossa

1. Thrombosis of the cavernous sinus, phlebitis and injury
2. Aneurysm
3. Herpes zoster
4. Infections of the paranasal sinuses
5. Tumor of the ridge of the sphenoid bone (meningioma, osteoma)
6. Other space-taking lesions in the middle fossa

Lesions of the Third Nerve in the Interpeduncular Space and at the Base of the Brain

1. Inflammatory lesions (acute and chronic meningitis; Weber's syndrome; interpeduncular syndrome)
2. Vascular lesions (aneurysms; sclerosis; subarachnoid hemorrhage; intraneural hemorrhage)
3. Tumor
4. Trauma

Lesions of the Third Nerve in the Region of the Midbrain

1. Nuclear Lesions
   (*a*) Acute infections (encephalitis; polioencephalitis; botulism; mushroom poisoning)
   (*b*) Wernicke's superior polioencephalitis
   (*c*) Vascular (diabetes; arteriosclerosis)
   (*d*) Tumor of the midbrain, tumor of the pons and tumor about the third ventricle
   (*e*) Intoxication (lead; carbon monoxide; barbiturates)
   (*f*) Progressive muscular atrophy
   (*g*) Chronic progressive ophthalmoplegia (von Graefe's)
   (*h*) Gerlier's disease, or kubisagari
   (*i*) Aphasia
2. Benedict's syndrome
3. Weber's syndrome

Supranuclear Lesions Affecting the Third Nerve

1. Cortical lesions (paralytic; irritative)
2. Pontile lesions (paralysis of lateral gaze with preservation of convergence)
3. Lesions of the midbrain
   Paralysis of upward movements (Parinaud's)
   Paralysis of downward movements
   Paralysis of convergence
   Vertical nystagmus

Miscellaneous Diseases of the Third Nerve

1. Pupillary abnormalities
   (*a*) The unilateral fixed dilated pupil in expanding cerebral lesions
   (*b*) Argyll Robertson pupil (syphilis; alcoholism; encephalitis; tumor in or about the midbrain; diabetes; multiple sclerosis; trauma)
   (*c*) Adie's syndrome
   (*d*) Other pupillary changes
2. Neuritis (diphtheritic and other systemic infections; "rheumatic neuritis"; neuritis due to serum treatment)
3. Arteriosclerosis and diabetes
4. Migrainous ophthalmoplegia
5. Periodic oculomotor paralysis (familial types)

DISCUSSION

DR. ROBERT A. GROFF: Dr. Yaskin's presentation is so complete that I have little to add. His paper is interesting, particularly from the anatomic point of view. From the surgical standpoint, I have been interested in the third nerve in connection with meningiomas of the ridge of the sphenoid bone. I have seen about 4 meningiomas of the lesser wing of the ridge, and in each instance there has been complete paralysis of the third nerve. In the case mentioned by Dr. Adler the

diagnosis was apparently made very early; he is to be congratulated on this. I have observed that in the cases in which there is complete palsy, even though the tumor is completely removed, there is no recovery from the palsy of the third nerve. I have also observed cases in which a pontile glioma or a pinealoma caused paralysis of upward or lateral gaze, which is the result of impairment of the third nerve. I have been interested in dilatation of the pupil occurring in association with subdural hematoma. In a series of over 50 cases, I observed 12 in which the pupil was dilated; in 6 of these the dilatation was observed on the same side as the hematoma and in 6 on the opposite side; this particular sign does not seem to have much value in localization. After the operation, in each instance, the dilatation of the pupil disappeared.

FRANCIS HEED ADLER, M.D., *Chairman*

WARREN S. REESE, M.D., *Clerk*

Jan. 16, 1941

A Case of Chiasmal Lesion. DR. WALTER I. LILLIE.

A white man aged 24 with intermittent symptoms of increased intracranial pressure of two months' duration was admitted to the Temple University Hospital. One month prior to admission there had suddenly developed bitemporal hemianopia and diplopia. General examination disclosed a bundle branch block associated with an enlarged heart and a mitral valvular lesion. Neurologic examination, performed by Dr. Temple Fay, revealed minor reflex disturbances, and the evidence pointed toward a diffuse basal lesion, either inflammatory or vascular, or to an aneurysm of the anterior communicating artery. Roentgenograms of the skull were normal. Ophthalmologic examination revealed that the vision was reduced to 6/30 in each eye. The upward ocular rotations were unevenly limited, with vertical diplopia. The perimetric fields revealed complete bitemporal hemianopia with bilateral central scotomas. Examination of the ocular fundi revealed no abnormality.

The spinal fluid pressure was increased to 24 mm. of mercury and dropped rapidly with the loss of 6 cc. of the fluid. The Wassermann reaction of the spinal fluid was negative, and the colloidal gold curve revealed no abnormality. Three days after drainage the vision in the left eye improved to 6/10 and the central scotoma in that eye disappeared. The right field of vision was unchanged.

Special studies for bruit gave negative results. In an attempt to establish the differential diagnosis between arachnoiditis and aneurysm and thus to determine whether surgical intervention was indicated, an encephalogram was requested at the neuro-ophthalmo-roentgenologic conference; this also revealed no abnormality. An aneurysm of the

anterior communicating artery ruptured suddenly five hours after the taking of the encephalogram, and the patient died. The diagnosis was confirmed at autopsy.

This case is presented to show that an aneurysm about the optic chiasm may produce signs and symptoms which may be more suggestive of acute basal arachnoiditis than of aneurysm. It demonstrates that all the available diagnostic procedures are necessary to determine whether surgical intervention should be advised. In this case, had the patient lived the encephalogram would have ruled out the necessity of surgical intervention. Aneurysms of this type are not operable.

Recent Progress in Central Field Studies. Dr. John N. Evans.

(By way of introduction it was pointed out that even the Hippocratic school considered the possible participation of blood vessels in the formation of defects of the visual field.

The basic anatomic and physiologic evidence which constitutes the rationale of angioscotometry was presented. The morphologic and topographic features of the angioscotoma were described, and were presented to show the physiologic variations of the defect. Examples of a wide variety of pathologic studies were presented, their features and significance explained and their usefulness in routine clinical work stressed.

The simple and rapid technic was emphasized, to contradict the erroneous impression that the work is complicated and tedious. In closing his presentation the speaker showed a tabulation of angioscotometric studies by workers in foreign lands and in the United States. In addition to being represented in more than thirty original contributions, angioscotometry is now discussed in all the standard textbooks on the visual fields and occupies increasing space in the current literature.)

Book Reviews

Extra-Ocular Muscles. By Luther C. Peter. Third edition. Price, $4.50. Pp. 368, with 147 engravings and 5 colored plates. Philadelphia: Lea & Febiger, 1941.

The third edition of this well known textbook on ocular motility appears just five years after the second edition, and although the words "thoroughly revised" appear on the title page, only 25 pages are either new or largely rewritten. The edition is otherwise a reprint of the second with a few unimportant changes.

One of the new sections deals with the technic of orthoptic training. No modern textbook on the treatment of strabismus is complete without a thorough discussion of this subject, but the 12 pages devoted to it in this book fail to accomplish that. The section starts with a definition of orthoptic exercises from Webster (!) and goes on to a discussion of "regular noncorrespondence," "false macula," "irregular or incongruous noncorrespondence" and "false projection." The subject of orthoptics is not so new that it has not been defined by competent authorities, and a well recognized terminology has been established for some of the newer concepts of binocular function; so it is difficult to find any justification for the introduction of these new terms. The author ignores the concept of abnormal retinal correspondence so ably set forth by Travers, Lyle and many others and substitutes four types of "noncorrespondence" that do not agree with the experience of most of the workers in this field. This entire section is written with such vagueness and lack of conviction that it can only confuse the student, whom the author professes to aid.

The chapter on nystagmus has been partially rewritten with the help of Joseph C. Yaskin, professor of neurology in the Graduate School of Medicine, University of Pennsylvania. Experimental forms of nystagmus are described and the results of the usual clinical tests given. This is of more interest to neurologists and otologists than to ophthalmologists but is a worth while addition to a book of this type. A detailed enumeration of the features to look for in a case of nystagmus and a table differentiating the three types of spontaneous nystagmus should be of practical value. The discussion of these three types (ocular, vestibular and neurologic) that follows would seem to clarify this difficult subject as much as is possible and is a distinct contribution. There is a bibliography at the end of the chapter.

At the end of the book the author tells briefly why he prefers to correct ptosis by shortening the levator muscle or by utilizing the occipitofrontalis muscle when the levator is completely paralyzed. He also tells how he reattaches the tenotomized muscle after an operation for retinal separation. One is so unaccustomed to look for this sort of information in such a book that I fear it will be overlooked.

It is regrettable that Peter does not point out the deficiencies of the Maddox rod and wing test for lateral deviations and that he still fails to emphasize that lateral deviations arise largely from anomalies of

convergence and divergence on an innervational basis, as was shown by Duane and has been accepted by increasing numbers of ophthalmologists. He maintains his stand that an alternating strabismus is one in which there is a complete absence of fusion, although there is ample evidence to the contrary.

This book has been set in a new type face that is definitely more pleasing, yet a number of the halftone illustrations are noticeably less distinct than in the previous edition. With the exception of the chapter on nystagmus, the few changes in this edition are of dubious value; so one questions the need for a new volume.

MAYNARD C. WHEELER.

CORRECTIONS

In the article by Dr. Walter F. Duggan entitled "Acute Retrobulbar Neuritis as a Manifestation of Acute Localized Tissue Anoxia: Treatment with Vasodilators," in the February issue (ARCH. OPHTH. **25:** 299, 1941), in the fourth and eighth lines of the last paragraph on page 307 "column 5" should be substituted for "column 7." On page 311, in the right hand column, for case 16, "sclerosis" should be substituted for "stenosis."

In the article by Dr. Walter E. Dandy entitled "Results Following the Transcranial Operative Attack on Orbital Tumors," in the February issue (ARCH. OPHTH. **25:** 191, 1941), in the last paragraph on page 213 the figure for the operative risk should be 4.1 per cent instead of 41 per cent.

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International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Consolacao, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive.
All correspondence should be addressed to the President.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7°.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 Ill tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Alfenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Albert C. Snell, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary: Dr. Derrick Vail, 441 Vine St., Cincinnati.
Place: Cleveland. Time: June 2-6, 1941.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. F. P. Calhoun, 478 Peachtree St. N. E., Atlanta, Ga.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va. Time: May 29-31, 1941.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. John Evans, 23 Schermerhorn St., Brooklyn.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. J. Vaillancourt, 46 St. Louis St., Quebec.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles W. Barkhorn, 223 Roseville Ave., Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. V. May, 1703 Main St., Marinette.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield
Place: Wisconsin Rapids. Time: June 1941.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Rowland, 84 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles, Calif.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Spencer S. Howe, 103 E. Holly St., Bellingham, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 419 Stimson Bldg., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. E. Rideout, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. Kessler, 311 Center Ave., Bay City, Mich.
Secretary-Treasurer: Dr. J. H. Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Davis, 1615 Howard St., Omaha.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Franklin P. Maury, Professional Bldg., Tucson, Ariz.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. J. K. Heckert, 103 N. Washington St., Lansing.
Secretary-Treasurer: Dr. W. D. Irwin, 710 Hanselman Bldg., Kalamazoo.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. H. Bailey, 29 S. Oakland Ave., Sharon.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. Raymond C. Cook, 701 Main St., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. William M. Bane, 1612 Tremont Pl., Denver.
Secretary: Dr. Harry Shankel, Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. W. E. McClellan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.
Place: Macon. Time: May 13-17, 1941.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. K. Von Lackum, 117–3d St. S.E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Henry N. Blum, 912 American Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Erling Wilhelm Hansen, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. George E. McGearv, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. A. L. Hammerel, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Edgar P. Cardwell, 47 Central Ave., Newark.
Secretary: Dr. Arthur E. Sherman, 243 S. Harrison St., East Orange.
Place: Atlantic City. Time: June 3-5, 1941.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Searle B. Marlow, 109 S. Warren St., Syracuse.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Secretary: Dr. Vanderbilt F. Couch, 105 W. 4th St., Winston-Salem.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Roderick Macdonald, Rock Hill.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia Hotel, Columbia. Time: November 1941.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. E. L. Goar, 1300 Walker Ave., Houston.
Secretary: Dr. Dan Brannin, 929 Medical Arts Bldg., Dallas.
Place: San Antonio. Time: December 1941.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Earl H. Phillips, 628 Judge Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. George G. Hawkins, Newport News.
Secretary-Treasurer: Dr. Guy Fisher, 3 E. Beverley St., Staunton.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Black, 7 W. Bowery St., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Stacy C. Howell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Maurice Wieselthier, 1322 Union St., Brooklyn.
Secretary-Treasurer: Dr. Harold F. Schilback, 142 Joralemon St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Richard Gamble, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. Josef Weintraub, 630 Vine St., Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert D. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Francis H. Adler, 313 S. 17th St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. D. Postle, 240 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. A. E. Jackson, 602 W. 10th St., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Wallace W. Ralston, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. John I. Garret, 57 Stokes Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Homer Beal, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Desmond Curran, Federal Reserve Bank Bldg., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Paul Southgate, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kirt G. Parks, 605 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Harold Mulligan, 1680 N. Vine St., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. E. J. Cummings, 1835 I St. N. W., Washington.
Secretary: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Guy Maness, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Leonard C. Whiting, 121 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Algernon Reese, 73 E. 71st St., New York.
Secretary: Dr. Brittain Payne, 896 Madison Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Morris Davidson, 80 Centre St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Leatherock, Cushing, Okla.
Secretary: Dr. Harry C. Ford, 1014 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. Howard Heine, 635 N. Main St., Fremont, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. L. Markowitz, 16 Church St., Paterson, N. J.
Secretary-Treasurer: Dr. A. John Reinhorn, 302 Broadway, Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Secretary: Dr. Wilfred E. Fry, 1819 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward A. Weisser, 119–5th Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. F. Bryce, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. E. J. Avery, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 5 p. m., Jan. 22. 1941.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. J. F. Hardesty, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fred Boyle, 490 Post St., San Francisco.
Secretary: Dr. Frank Hand, 450 Sutter St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Louis A. Parsell, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. F. R. Webster, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. John R. Myers, State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. S. H. Patterson, 1251–15th St., Toledo, Ohio.
Secretary: Dr. J. E. Minns, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. Frank Costenbader, 1726 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. L. Conner Moss, 1710 Rhode Island Ave. N.W., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.